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RESPIRATORY CARE

A MONTHLY SCIENCE JOURNAL
37TH YEAR—ESTABLISHED 1956

SPECIAL ISSUE
Conference on
the Essentials of
Mechanical Ventilators

- Consensus Statement on the Essentials of Mechanical Ventilators—1992
- Classification of Mechanical Ventilators
- Technical Description and Classification of Modes of Ventilator Operation
- Essential Gas Delivery Features of Mechanical Ventilators
- Mechanical Ventilator Design and Function: The Trigger Variable
- Inspired Gas Conditioning
- Monitoring Pressure, Flow, and Volume during Mechanical Ventilation
- What Derived Variables Should Be Monitored during Mechanical Ventilation?
- Essentials for Ventilator-Alarm Systems
- Digital Electronic Communication between ICU Ventilators and Computers and Printers
- What Constitutes an Order for Mechanical Ventilation, and Who Should Give the Order?

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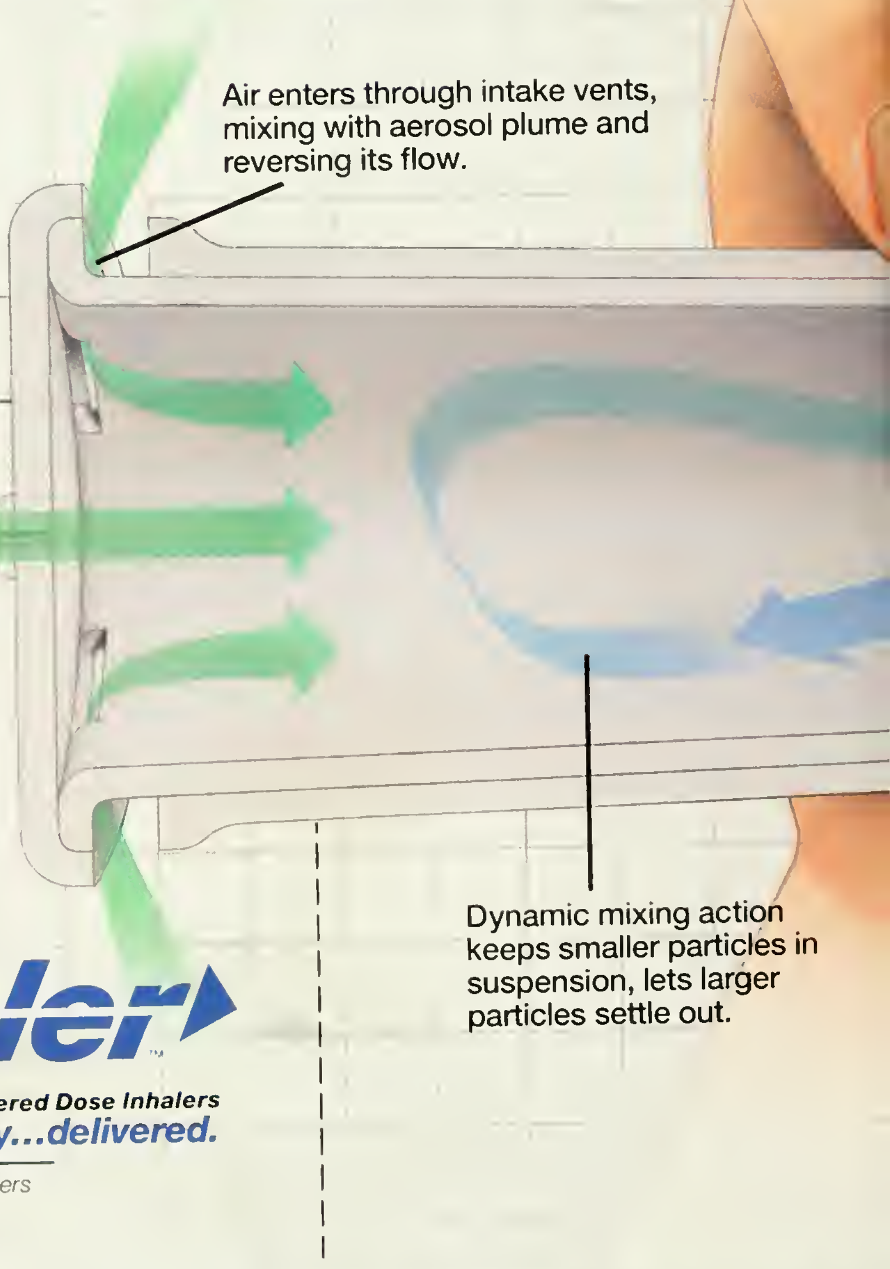

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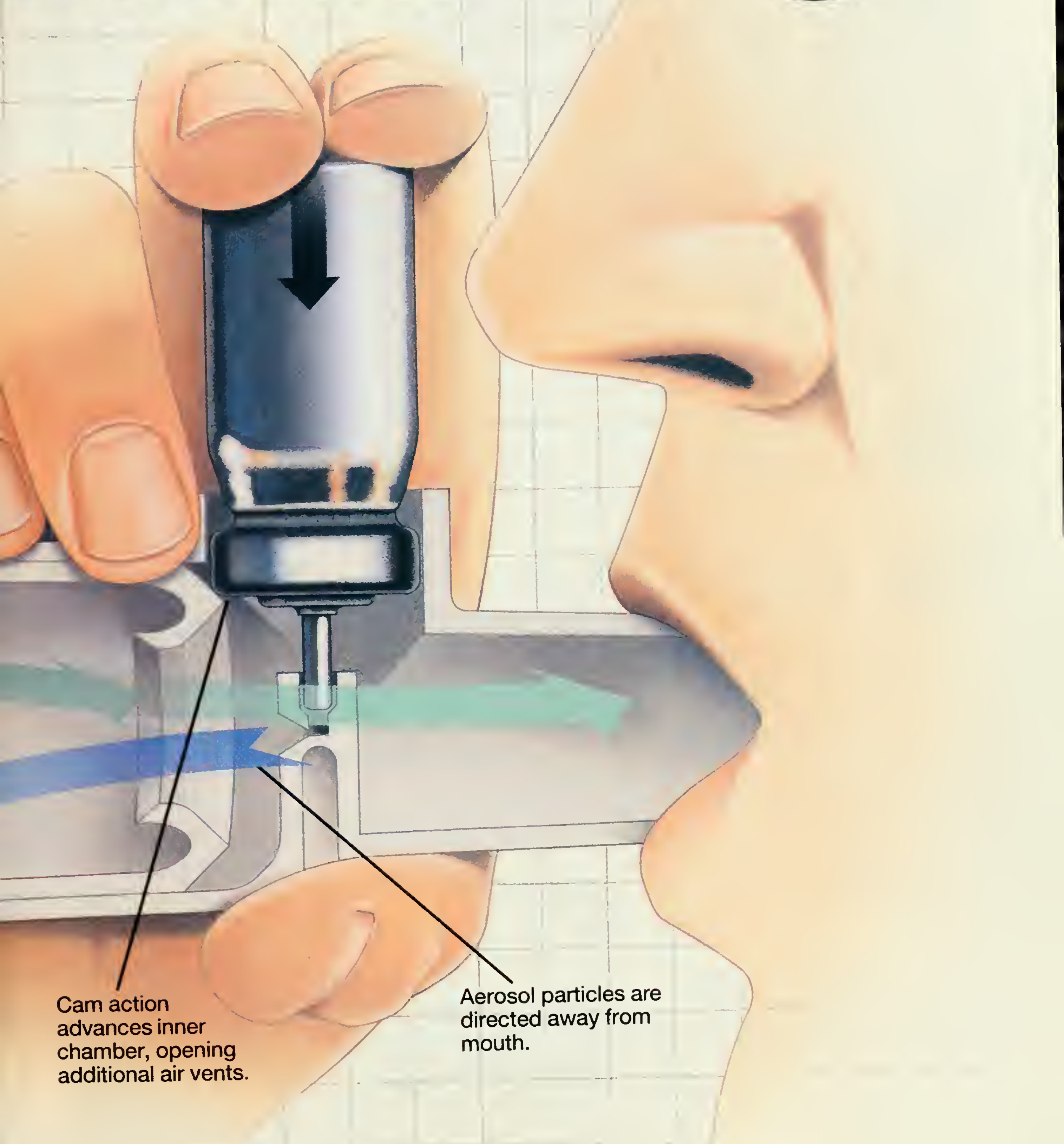
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References: 1-2. Data on file, HealthScan Products Inc. U.S. Patent No. 5,040,527 OA760002-0 8/92

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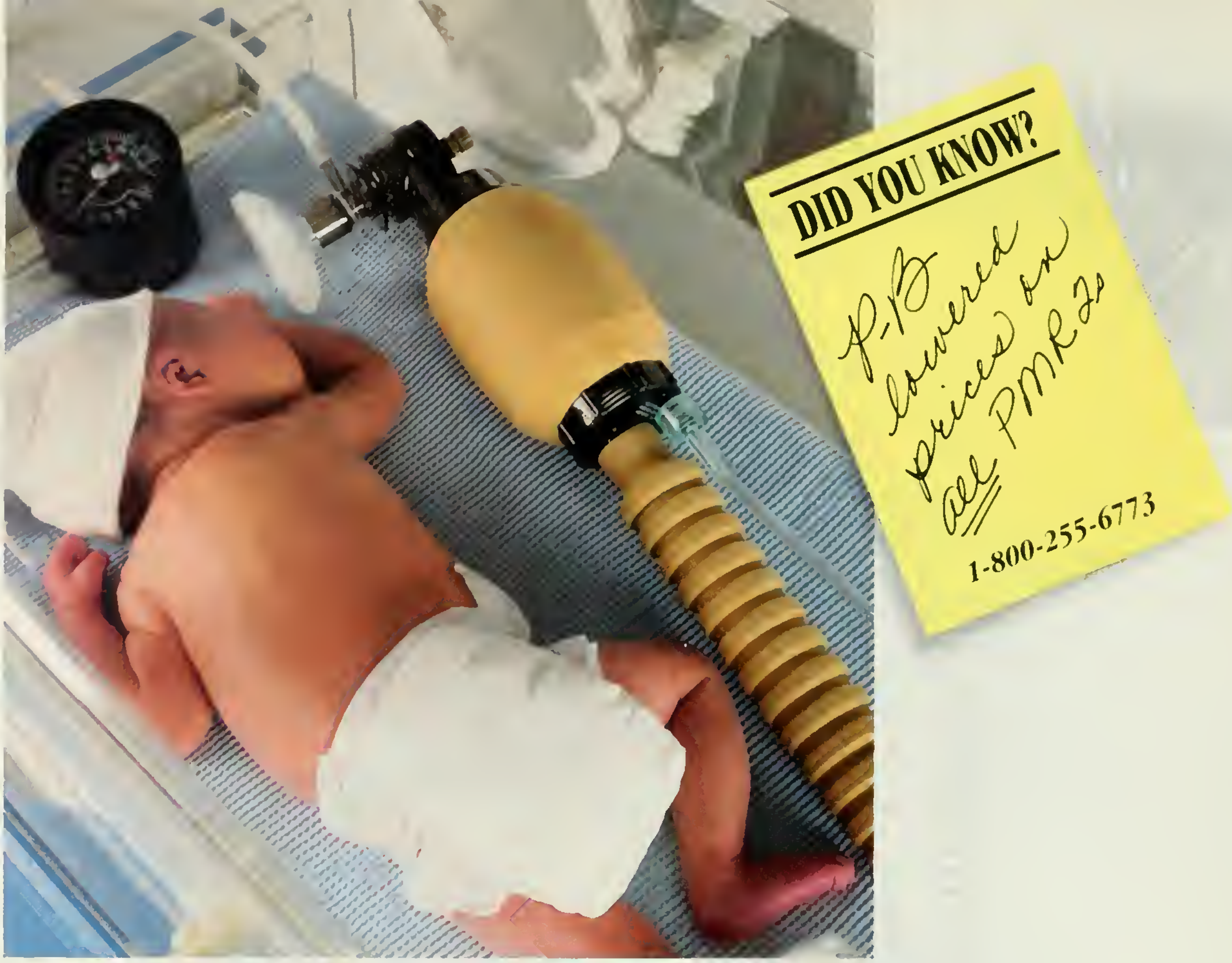

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SPECIAL ISSUE

CONSENSUS CONFERENCE ON THE ESSENTIALS OF MECHANICAL VENTILATORS

The Proceedings of a Conference
held February 27-29, 1992,
in Cancun, Mexico

Neil R MacIntyre MD and Richard D Branson RRT
Chairmen and Guest Editors

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Hand-Washing and Nosocomial Infections (editorial)—D Goldmann, E Larson. N Engl J Med 1992;327:120. (Pertains to Doebbeling et al paper abstracted below.)

Airway Responsiveness: New Concepts and Concerns (editorial)—GL Larsen. Pediatr Pulmonol 1992;13:69.

Bronchopulmonary Dysplasia: Twenty-Five Years Later (commentary)—WH Northway Jr. Pediatrics 1992;89:969.

Resuscitation and Withdrawal of Therapy in Pediatric Intensive Care (brief report)—RB Mink, MM Pollack. Pediatrics 1992;89:961.

Re-Targeting Ventilatory Objectives in Adult Respiratory Distress Syndrome: New Treatment Prospects—Persistent Questions (editorial)—JJ Marini, SG Kelsen. Am Rev Respir Dis 1992;146:2-3.

Comparative Efficacy of Alternative Hand-Washing Agents in Reducing Nosocomial Infections in Intensive Care Unit—BN Doebbeling, GL Stanley, CT Sheetz, MA Pealler, AK Houston, L A Ning Li, RP Wenzel 1992;327:88.

BACKGROUND: Effective hand-washing can prevent nosocomial infections, particularly in high risk areas of the hospital. There are few clinical studies of the efficacy of specific hand-cleansing agents in preventing the transmission of pathogens from health care workers to patients. METHODS: For 8 months, we conducted a prospective multiple-crossover trial involving 1,894 adult patients in three intensive care units (ICUs). In a given month, the ICU used a hand-washing system involving either chlorhexidine, a broad-spectrum antimicrobial agent, or 60% isopropyl alcohol with the optional use of a nonmedicated soap; in alternate months the other system was used. Rates of nosocomial infection and hand-washing compliance were monitored prospectively. RESULTS: When chlorhexidine was used, there were 152 nosocomial infections, as compared with 202 when the combination of alcohol and soap was used (adjusted incidence-density ratio [IDR], 0.73; 95% confidence interval, 0.59-0.90). The largest reduction with chlorhexidine was in gastrointestinal infections (IDR, 0.19; 95% confidence interval, 0.05-0.64). When chlorhexidine was available, the rates of nosocomial infection declined in each of the ICUs, and health care workers washed their hands more often than when alcohol and soap were used (relative risk, 1.28; 95% confidence interval, 1.02-1.60). The total volume of alcohol and soap used was 46% that of chlorhexidine ($p < 0.001$). CONCLUSIONS: A hand-disinfection system using an antimicrobial agent (chlorhexidine) reduces the rate of nosocomial infections more effectively than one using alcohol and soap. The improvement may be explained at least in part by better compliance with hand-washing instructions when chlorhexidine was used.

Safety of Bronchoalveolar Lavage and Bronchial Biopsies in Patients with Asthma of Variable Severity—T Van Vyve, P Chanez, J Bousquet, J-Y Lacoste, F-B Michel, P Godard. Am Rev Respir Dis 1992; 146:116.

The safety of fiberoptic bronchoscopy, bronchoalveolar lavage (BAL), and bronchial biopsies has been questioned in asthma, and current

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In premature infants less than 1250 g birth weight or with evidence of surfactant deficiency, give SURVANTA as soon as possible, preferably within 15 minutes of birth.

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To treat infants with RDS confirmed by x-ray and requiring mechanical ventilation, give SURVANTA as soon as possible, preferably by 8 hours of age.

CONTRAINDICATIONS

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WARNINGS

SURVANTA is intended for intratracheal use only.

SURVANTA CAN RAPIDLY AFFECT OXYGENATION AND LUNG COMPLIANCE. Therefore, its use should be restricted to a highly supervised clinical setting with immediate availability of clinicians experienced with intubation, ventilator management, and general care of premature infants. Infants receiving SURVANTA should be frequently monitored with arterial or transcutaneous measurement of systemic oxygen and carbon dioxide.

DURING THE DOSING PROCEDURE, TRANSIENT EPISODES OF BRADYCARDIA AND DECREASED OXYGEN SATURATION HAVE BEEN REPORTED. If these occur, stop the dosing procedure and initiate appropriate measures to alleviate the condition. After stabilization, resume the dosing procedure.

PRECAUTIONS

General

Rales and moist breath sounds can occur transiently after administration. Endotracheal suctioning or other remedial action is not necessary unless clear-cut signs of airway obstruction are present.

Increased probability of post-treatment nosocomial sepsis in SURVANTA-treated infants was observed in the controlled clinical trials (Table 3). The increased risk for sepsis among SURVANTA-treated infants was not associated with increased mortality among these infants. The causative organisms were similar in treated and control infants. There was no significant difference between groups in the rate of post-treatment infections other than sepsis.

Use of SURVANTA in infants less than 600 g birth weight or greater than 1750 g birth weight has not been evaluated in controlled trials. There is no controlled experience with use of SURVANTA in conjunction with experimental therapies for RDS (eg, high-frequency ventilation or extracorporeal membrane oxygenation).

No information is available on the effects of doses other than 100 mg phospholipids/kg, more than four doses, dosing more frequently than every 6 hours, or administration after 48 hours of age.

Carcinogenesis, Mutagenesis, Impairment of Fertility

Reproduction studies in animals have not been completed. Mutagenicity studies were negative. Carcinogenicity studies have not been performed with SURVANTA.

ADVERSE REACTIONS

The most commonly reported adverse experiences were associated with the dosing procedure. In the multiple-dose controlled clinical trials, transient bradycardia occurred with 11.9% of *doses*. Oxygen desaturation occurred with 9.8% of *doses*.

Other reactions during the dosing procedure occurred with fewer than 1% of doses and included endotracheal tube reflux, pallor, vasoconstriction, hypotension, endotracheal tube blockage, hypertension, hypocarbia, hypercarbia, and apnea. No deaths occurred during the dosing procedure, and all reactions resolved with symptomatic treatment.

The occurrence of concurrent illnesses common in premature infants was evaluated in the controlled trials. The rates in all controlled studies are in Table 3.

TABLE 3

	All Controlled Studies		
Concurrent Event	**SURVANTA (%)**	**Control (%)**	***P*-Value**[a]
Patent ductus arteriosus	46.9	47.1	0.814
Intracranial hemorrhage	48.1	45.2	0.241
Severe intracranial hemorrhage	24.1	23.3	0.693
Pulmonary air leaks	10.9	24.7	<0.001
Pulmonary interstitial emphysema	20.2	38.4	<0.001
Necrotizing enterocolitis	6.1	5.3	0.427
Apnea	65.4	59.6	0.283
Severe apnea	46.1	42.5	0.114
Post-treatment sepsis	20.7	16.1	0.019
Post-treatment infection	10.2	9.1	0.345
Pulmonary hemorrhage	7.2	5.3	0.166

[a] *P*-value comparing groups in controlled studies

When all controlled studies were pooled, there was no difference in intracranial hemorrhage. However, in one of the single-dose rescue studies and one of the multiple-dose prevention studies, the rate of intracranial hemorrhage was significantly higher in SURVANTA patients than control patients (63.3% *v* 30.8%, *P* = 0.001, and 48.8% *v* 34.2%, *P* = 0.047, respectively). The rate in a Treatment IND involving approximately 4400 infants was lower than in the controlled trials.

In the controlled clinical trials, there was no effect of SURVANTA on results of common laboratory tests: white blood cell count and serum sodium, potassium, bilirubin, creatinine.

More than 3700 pretreatment and post-treatment serum samples were tested by Western Blot immunoassay for antibodies to surfactant-associated proteins SP-B and SP-C. No IgG or IgM antibodies were detected.

Several other complications are known to occur in premature infants. The following conditions were reported in the controlled clinical studies. The rates of the complications were not different in treated and control infants, and none of the complications were attributed to SURVANTA.

Respiratory: lung consolidation, blood from the endotracheal tube, deterioration after weaning, respiratory decompensation, subglottic stenosis, paralyzed diaphragm, respiratory failure.

Cardiovascular: hypotension, hypertension, tachycardia, ventricular tachycardia, aortic thrombosis, cardiac failure, cardio-respiratory arrest, increased apical pulse, persistent fetal circulation, air embolism, total anomalous pulmonary venous return.

Gastrointestinal: abdominal distention, hemorrhage, intestinal perforations, volvulus, bowel infarct, feeding intolerance, hepatic failure, stress ulcer.

Renal: renal failure, hematuria.

Hematologic: coagulopathy, thrombocytopenia, disseminated intravascular coagulation.

Central Nervous System: seizures.

Endocrine/Metabolic: adrenal hemorrhage, inappropriate ADH secretion, hyperphosphatemia.

Musculoskeletal: inguinal hernia.

Systemic: fever, deterioration.

Follow-Up Evaluations

To date, no long-term complications or sequelae of SURVANTA therapy have been found.

Single-Dose Studies

Six-month adjusted-age follow-up evaluations of 232 infants (115 treated) demonstrated no clinically important differences between treatment groups in pulmonary and neurologic sequelae, incidence or severity of retinopathy of prematurity, rehospitalizations, growth, or allergic manifestations.

Multiple-Dose Studies

Six-month adjusted age follow-up evaluations have not been completed. Preliminarily, in 605 (333 treated) of 916 surviving infants, there are trends for decreased cerebral palsy and need for supplemental oxygen in SURVANTA infants. Wheezing at the time of examination tended to be more frequent among SURVANTA infants, although there was no difference in bronchodilator therapy.

Twelve-month follow-up data from the multiple-dose studies have been completed in 328 (171 treated) of 909 surviving infants. To date no significant differences between treatments have been found, although there is a trend toward less wheezing in SURVANTA infants in contrast to the six month results.

OVERDOSAGE

Overdosage with SURVANTA has not been reported. Based on animal data, overdosage might result in acute airway obstruction. Treatment should be symptomatic and supportive.

Rales and moist breath sounds can transiently occur after SURVANTA is given, and do not indicate overdosage. Endotracheal suctioning or other remedial action is not required unless clear-cut signs of airway obstruction are present.

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SURVANTA (beractant) Intratracheal Suspension is supplied in single-use glass vials containing 8 mL of SURVANTA (NDC 0074-1040-08). Each milliliter contains 25 mg of phospholipids (200 mg phospholipids/8 mL) suspended in 0.9% sodium chloride solution. The color is off-white to light brown.

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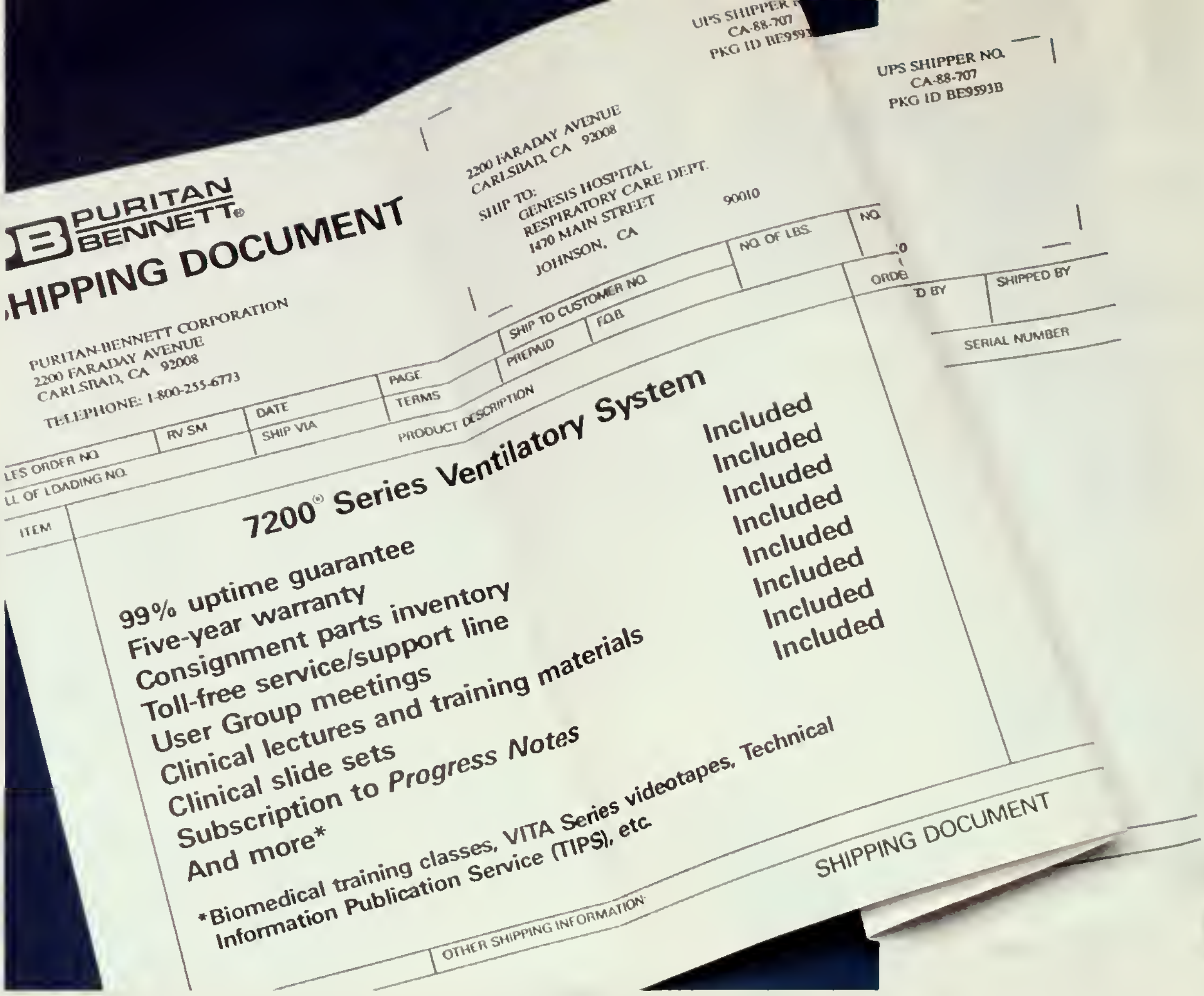
recommendations indicate that bronchoscopies should only be performed in mild to moderate asthma. Moreover, in most studies patients receive premedication with nebulized bronchodilators that may enhance the safety of the procedures. The purpose of this study was to determine (1) whether the overall safety of fiberoptic bronchoscopy, BAL, and bronchial biopsies in mild to moderate asthma could be extended to patients with more severe asthma and (2) whether these procedures are safe without premedication with nebulized bronchodilators. A group of 50 patients with asthma of variable severity (FEV_1, ranging from 37-107% of predicted values) and 25 healthy volunteers were studied. Bronchoscopy, BAL (250 mL), and four bronchial biopsies were performed in a standardized manner, without premedication with a nebulized bronchodilator, by the same investigator. Safety was assessed by clinical follow-up, continuous recording of arterial oxygen saturation during the procedure with a digital oximeter, and measuring FEV_1, $FEF_{25\text{-}75}$, and FVC just before and 5 min after bronchoscopy. Arterial oxygen saturation decreased in asthmatic patients from 97% (range 91-99%) (T1) to 92% (range 79-98%) (T8) (ANOVA, Fisher's PLSD) and in control subjects from 97% (range 94-99%) (T1) to 93% (range 88-98%) (T8) (ANOVA, Fishers PLSD). The fall in arterial oxygen saturation was not significantly different between asthmatic and normal subjects, and there was no correlation between arterial oxygen desaturation and the severity of asthma. In asthma, FVC decreased significantly from 86.2 ± 14.6 to 64.0 ± 17.1% ($p = 0.0001$) and FEV_1 decreased significantly from 75.6 ± 16.8 to 55.3 ± 17.2% ($p = 0.0002$). The fall in FEV_1 was not related to the severity of asthma. In control subjects, FVC decreased significantly from 99.6 ± 14.3 to 82.3 ± 19.0% ($p = 0.0125$) and FEV_1 decreased significantly from 97.1 ± 14.0 to 80.3 ± 16.2% ($p = 0.0071$). The percentages of falls in FVC, FEV_1, and $FEF_{25\text{-}75}$ after endoscopic procedure were significantly greater in asthmatic than in control subjects ($p = 0.0121$, $p = 0.0124$, and $p = 0.0217$, respectively). There were no significant falls in the ratios FEV_1/FVC or $FEF_{25\text{-}75}$/FVC, either in asthmatic or in control subjects. BAL and biopsies are well tolerated in asthmatic patients without premedication with a nebullized bronchodilator, even if they have severe asthma or a low FEV_1 before bronchoscopy.

Effect of Allergen Avoidance on Development of Allergic Disorders in Infancy—SH Arshad, S Matthews, C Gant, DW Hide. Lancet 1992;339:1493.

There is much evidence that the development of allergic disorders may be related to early exposure of allergens, including those in breastmilk. We have tried to find out whether avoidance of food and inhaled allergens in infancy protects against the development of allergic disorders in high-risk infants. In a prenatally randomised, controlled study 120 infants with family history of atopy and high (> 0.5 kU/L) cord-blood concentrations of total IgE were allocated randomly to prophylactic and control groups. In the prophylactic group ($n = 58$), lactating mothers avoided allergenic foods (milk, egg, fish, and nuts) and avoided feeding their infants these foods and soya, wheat, and orange up to the age of 12 months; the infants' bedrooms and living rooms were treated with an acaricidal powder and foam every 3 months, and concentrations of *Dermatophagoides pteronyssinus* antigen (*Der p 1*) in dust samples were measured by enzyme-linked immunosorbent assay. In the control group ($n = 62$), the diet of mothers and infants was unrestricted; no acaricidal treatment was done and *Der p 1* concentrations were measured at birth and at 9 mo. A pediatric allergy specialist unaware of group assignment examined the infants for allergic disorders at 10-12 mo. Odds ratios were calculated by logistic regression analysis for various factors with control for other confounding variables. At 12 mo, allergic disorders had developed in 25 (40%) control infants and in 8 (13%) of the prophylactic group (odds ratio 6.34, 95% confidence interval 2.0-20.1). The prevalences at 12 months of asthma (4.13, 1.1-15.5) and eczema (3.6, 1.0-12.5) were also significantly greater in the control group. Parental smoking was a significant risk factor for total allergy at 12 months whether only one parent smoked (3.97, 1.2-13.6) or both parents smoked (4.72, 1.2-18.2). Reduced exposure of infants to allergens in food and in housedust lowered the frequency of allergic disorders in the first years of life. Passive smoking is an important risk factor that should be addressed in any prophylactic programme.

Double-Blind Study of Selective Decontamination of the Digestive Tract in Intensive Care—JMJ Hammond, PD Potgieter, GL Saunders, AA Forder. Lancet 1992;340:5.

Selective decontamination of the digestive tract (SDD), by means of non-absorbable antibiotics, to prevent infection in intensive-care units (ICUs) remains controversial; there is evidence that the regimen reduces the incidence of secondary infection, but no convincing reduction in morbidity or mortality has been shown and the costs and effect on microbial resistance patterns need further study. In a double-blind, placebo-controlled trial, we have tried to find out whether SDD should be used



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routinely in all ICU patients at high risk of secondary infection. All patients admitted to the ICU who were thought likely to stay in the unit for at least 5 days and to need intubation for longer than 48 h were enrolled and randomly allocated to groups receiving placebo or SDD (amphotericin, colistin, and tobramycin applied to the oropharynx and enterally); all patients received intravenous cefotaxime for 72 h. Of 322 patients randomised, 83 were withdrawn (80 ICU stay or duration of intubation too short, 3 protocol violations). 239 medical, trauma, and surgical patients completed the trial period (114 SDD, 125 placebo). There were no differences between SDD and placebo groups in incidence of infection (30 [26%] vs 43 [34%] patients; p = 0.22), duration of ICU stay (mean 16.2 [14.3] vs 16.8 [12.3] days), hospital stay (29.9 [SD 25.0] vs 31.9 [22.2] days), or mortality (21 [18%] vs 21 [17%]). SDD substantially increased the costs of intensive care. Mechanisms other than bacterial colonisation of the gut may bring about substantial numbers of secondary infections in ICUs. Routine use of SDD in multidisciplinary ICUs cannot be recommended.

Economic Benefits of Teaching Patients with Chronic Obstructive Pulmonary Disease about Their Illness—L Tougaard, T Krone, A Sorknaes, H Ellegaard, and the PASTMA Group. Lancet 1992;339: 1517.

By instructing patients in how to deal with their disease, financial demands on health services may be reduced. 100 consecutive patients (aged 48-89) admitted to a general medical ward in Denmark with chronic obstructive pulmonary disease (COPD) were allocated randomly to receive either "personalised hospital practice" (PHP), which includes training in aspects of their disease, or standard hospital practice. Changes in "consumption" of health services per patient from 1 year before until 1 year after the intervention admission were evaluated in 82 (PHP group 42, controls 40) patients who completed the intervention phase. Each group contained about the same percentage of asthmatics and smokers. The increase in consumption of health services after intervention was on average Kr 15298 [1 Kr ≈ $0.18] per patient per year less in the PHP group than in the control group (p = 0.048, Wilcoxon test). Consumption of general practitioner services was significantly increased in the control group compared with the PHP group (mean [95% CI] Kr 1346 [549 to 2143] vs –89 [–423 to 245] per patient per year; p = 0.001, Wilcoxon test). These differences could not be explained by changes in smoking habits. PHP reduces the consumption of health services by patients with COPD, probably by increasing patients' knowledge of disease and hence their ability to manage themselves.

Intermediate-Term Results of Heart-Lung Transplantation for Cystic Fibrosis—BP Madden, ME Hodson, V Tsang, R Radley-Smith, A Khaghani, MY Yacoub. Lancet 1992;339:1583.

Between September, 1984, and March, 1991, 79 patients underwent heart-lung transplantation for end-stage cystic fibrosis at the Harefield Hospital. Short-term outcome has already been reported, and we now present intermediate-term results. The overall actuarial patient survival was 69% at 1 year, 52% at 2 years, and 49% at 3 years. 17 patients had diabetes mellitus with a survival of 62% to 1 year and 51% to 2 years. 23 patients had one or more other possible high-risk factors, and survival of these patients was 64% at 1 year and 57% at 2 years, compared with 71% and 49%, respectively, in the low-risk group (n = 56). *Pseudomonas aeruginosa* infection was the most common respiratory infection encountered postoperatively. 92% of patients had at least one episode of acute rejection during the first 3 postoperative months. Lung function was greatly improved after transplantation, the mean forced expiratory volume in 1 s and forced vital capacity increasing from 22% and 35% predicted, respectively, preoperatively to 68% and 70% predicted, respectively, by the sixth postoperative month. This improvement was maintained at 1, 2, and 3 years after transplantation. Lymphoproliferative disorders (4 patients) were successfully treated. Obliterative bronchiolitis developed in 17 patients and the cumulative probability of getting this complication at 1, 2, and 3 years postoperatively was 17%, 23%, and 48%, respectively. Overall, 7 patients were retransplanted. There was no coronary artery disease in the 37 patients who underwent coronary angiography at 1 year, 14 at 2 years, and 9 at 3 years after surgery. 58 patients donated their hearts for subsequent "domino" heart tranplantation. Our 5.5-year experience with heart-lung transplantation is encouraging, but the shortage of donor organs and the complication of obliterative bronchiolitis are the two main obstacles to be overcome.

Reproducibility of Visual Analog Scale Measurements of Dyspnea in Patients with Chronic Obstructive Pulmonary Disease—MJ Mador, TJ Kufel. Am Rev Respir Dis 1992;146: 82.

The purpose of this study was to evaluate the reproducibility of visual analog scale ratings of the effort to breathe (VAS_e) and the degree of discomfort evoked by breathing (VAS_d) in patients with chronic obstructive pulmonary disease (COPD)

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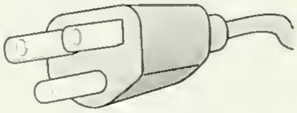

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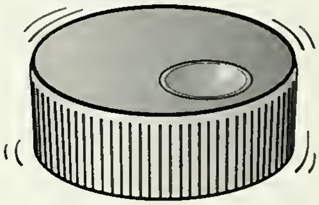

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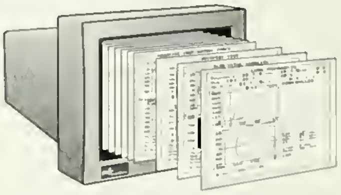

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during exercise. Six subjects with moderately severe COPD (FEV_1 = 1.12 + 0.29 L, FEV_1/FVC = 44 ± 4%) underwent progressive incremental exercise testing to a symptom-limited maximum every week for 8 wk. VAS_e and VAS_d were highly correlated in each subject ($r = 0.99 \pm 0.01$). The slope of the VAS_d/VAS_e relationship for all trials in all subjects was not significantly different from 1, indicating that our subjects were rating a common sensation with the two scales. VAS_e at maximal exercise was reproducible in every subject; the within-subject coefficient of variation (CV) was 6% (range 2-10%) and compared favorably with physiologic indices; 7% (range, 3-12%) for oxygen consumption and 10% (range 5-16%) for minute ventilation ($\dot{V}_I$). In contrast, submaximal VAS ratings were highly variable. At 66% of the maximal work load, the within-subject CV for VAS_e was 21% (range 11-28%) compared with 6% (range, 4-7%) for $\dot{V}_{O_2}$ ($p < 0.003$) and 10% (range, 5-16%) for $\dot{V}_I$ ($p < 0.01$). VAS_e correlated linearly with $\dot{V}_I$ and $\dot{V}_{O_2}$ in all subjects in all trials. However, within an individual subject the slope and position of these relationships varied widely between trials. We conclude that although maximal VAS ratings are reproducible, submaximal VAS ratings and the relationshlp between VAS ratings and physiologic indices vary considerably when exercise tests are performed at weekly intervals in patients with COPD.

Peripheral Airways Resistance in Smokers—EM Wagner, ER Bleecker, S Permutt, MC Liu. Am Rev Respir Dis 1992;146:92.

To determine peripheral airways resistance (R_P) in asymptomatic smokers ,we used a wedged bronchoscope technique to study 19 volunteers (18-44 yr of age) who actively smoked for 2 to 28 pack-years. A fiberoptic bronchoscope was wedged in a subsegmental bronchus of the right upper lobe. Using a double lumen catheter inserted through the working channel of the bronchoscope, we infused 5% CO_2 in air through one lumen and measured pressure through the second lumen. R_P was determined as the average of the peripheral resistance measured at three or more flowrates. This resistance ranged from 0.003-0.075 cm H_2O/mL/min in the 19 subjects. We have previously shown normal subjects to have an average R_P of 0.009 ± 0.002 cm H_2O/mL/min (mean ± SE) and asthmatic subjects an average of 0.069 ± 0.017 cm H_2O/mL/min. Thus, despite normal pulmonary function as assessed by spirometry, these asymptomatic smokers demonstrated a wide range of R_P values from normal to that observed in asthmatic subjects. These findings are consistent with a mechanism that considers the high resistance to result from inflammatory changes in the small airways.

Long-Term Prognosis of Near-Fatal Asthma: A 6-Year Follow-Up Study of 145 Asthmatic Patients Who Underwent Mechanical Ventilation for a Near-Fatal Attack of Asthma—CH Marquette, F Saulnier, O Leroy, B Wallaert, C Chopin, JM Demarcq, A Durocher, AB Tonnel. Am Rev Respir Dis 1992;146:76.

The objective of the present study was to investigate the long-term prognosis of near-fatal asthma. A retrospective cohort study design was used. Cases were defined as any asthmatic individual requiring mechanical ventilation for the first time for an asthma exacerbation between January 1, 1983 and December 31, 1988. The consecutive sample of patients was drawn from four study sites, specifically four intensive care units (ICU), based in a large urban area (1 million inhabitants). These four ICUs total approximately 5,000 admissions per year and are the referral centers for more than 95% of patients requiring respiratory intensive care in the area. Data collection was obtained by questionnaires adressed to the patients and to their attending physicians and was completed by telephone calls if necessary. A total of 147 patients entered the study. The long-term outcome could be evaluated in all but 2 patients. The follow-up period ranged from 1-75 mo. In-hospital mortality was 16.5%. Among the 121 patients discharged from the ICU, 18 subsequently died, 17 of whom died from a new attack of asthma. Posthospitalization mortality was 10.1% (95% CI, 5.9-16.8%) after 1 yr, 14.4% (CI, 9-22.3%) after 3 yr, and 22.6% (CI, 12.7-36.8%) after 6 yr. Nearly two-thirds (61.5%) of these secondary deaths occurred within the year following discharge from the ICU. Smoking was associated with a higher in-hospital mortality, as well as with a higher posthospitalization mortality. Age was also independently associated with a higher posthospitalization mortality. It is noteworthy that the secondary deaths were mostly observed in patients over 40 years of age. Smoking was associated with a significantly higher rehospitalization rate. The high prevalence of aspirin-sensitive asthma was of concern, but aspirin-sensitive asthma was not by itself related to a poorer prognosis. Specialized outpatient management such as regular survey by pulmonary physicians and assessment of pulmonary function, was surprisingly infrequent. The present study supports the evidence that smoking is associated with a higher mortality in some asthmatics and underscores the need for close and continuous surveillance of patients who have experienced a near-fatal attack of asthma.

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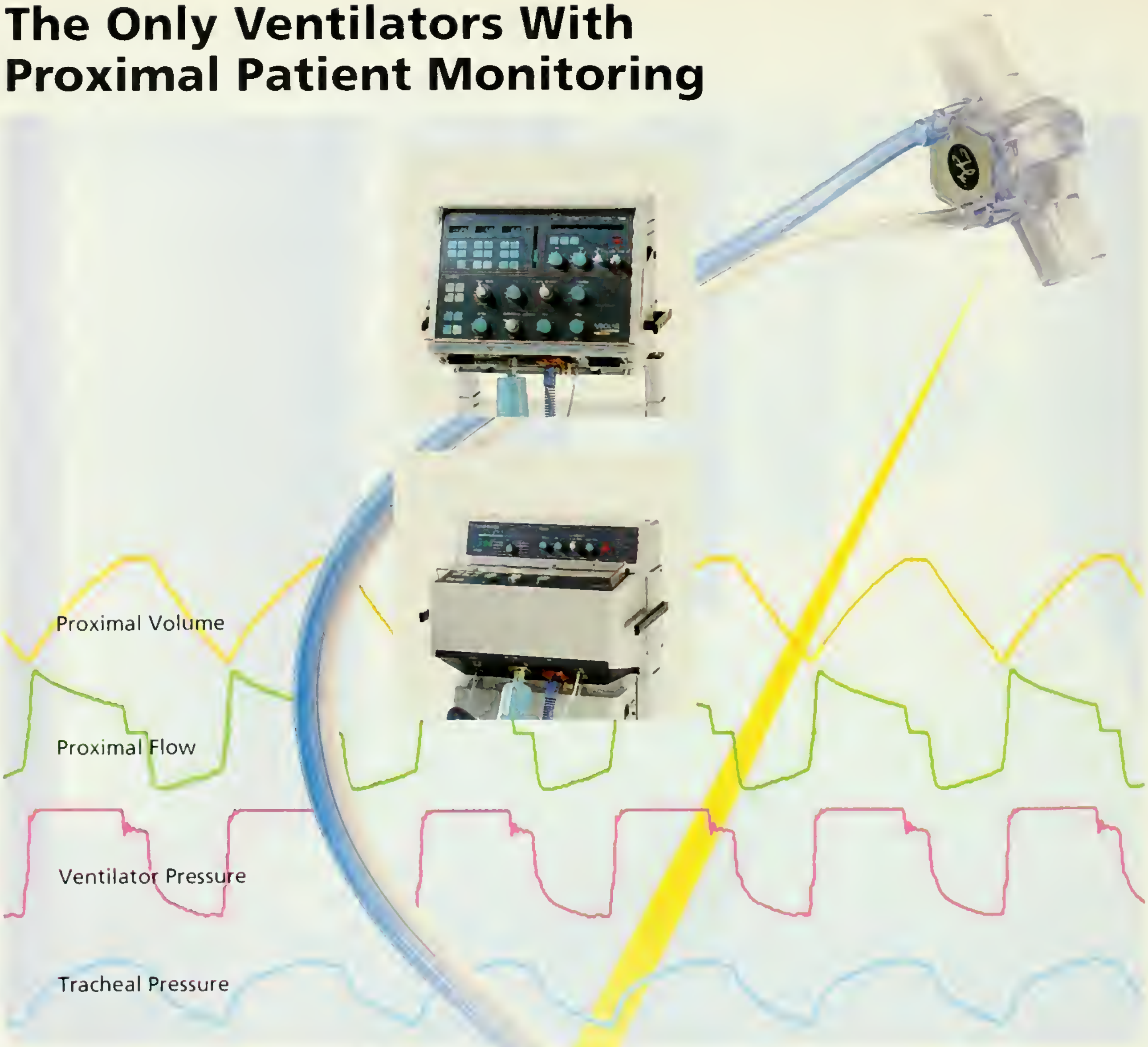

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Measurement of Particle and Mass Distribution of Pentamidine Aerosol by Ultrasonic and Air Jet Nebulizers—J Hager, K-H Gober, J-P Löhr, M Dürr. J Aerosol Med 1992: 5:65.

The use of appropriate nebulizers is a major precondition for a successful treatment and prevention of *Pneumocystis carinii* pneumonia with pentamidine aerosol. The apparatus should supply a sufficient amount of pentamidine with adequate particle size. Using Fisons ultrasonic nebulizer FISO Neb, Model FZV 40 BAMKI, DeVilbiss ultrasonic nebulizer, Porta-Sonic, Model 8500 GB, and the Marquest Medical Products jet nebulizer Respirgard II, two pentamidine concentrations (300 mg/6 mL and 60 mg/6 mL) were compared by measuring nebulized pentamidine mass distribution and particle-size distribution under in-vitro conditions by means of a laser light-scattering particle sizer of the type Malvern Master sizer. It was found that there were significant differences among nebulizers. Mass distribution experiments with airflow 6 L/min showed that using FISO Neb the quantity of nebulized pentamidine was 201.4 mg and 36.7 mg, whereas using Porta-Sonic the values found decreased to 85.2 mg and 23.6 mg. Using Respirgard II the values were 80.0 mg and 10.64 mg. The measured total duration times of nebulization were 6-8.5 min, 12 min, and 25 min for the nebulizers FISO Neb, Porta-Sonic, and Respirgard II. A decomposition of pentamidine during nebulization in the case of ultrasonic nebulizers doesn't take place. The measured mass median diameters (MMD) were 5.6-6.9 μm, 1.96-3.04 μm, and 1.9-2.5 μm for the nebulizers FISO Neb, Porta-Sonic and Respirgard II. Using 300 mg pentamidine the nebulized amounts of pentamidine containing particles sizes 2 μm predominately available for alveolar deposition were with (values of about 43 mg) markedly higher for Respirgard II and Porta-Sonic than the measured 10.5 mg for FISO Neb.

The Effect of Acid Aerosols on Airway Responsiveness in Asthmatics—R Jörres, H Magnussen. J Aerosol Med 1992;5:103.

Most investigations have shown that short-term exposure to acid aerosols did not cause airway obstruction in normal subjects at concentrations up

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to 1 mg/m^3 whereas some studies could demonstrate adverse effects in asthmatic subjects at concentrations of 0.1 mg/m^3 or higher. Inhalation of acid aerosols may produce airway hyperreactivity to carbachol in normal subjects and may enhance airway hyperreactivity in asthmatic subjects. We investigated 12 patients with bronchial asthma inhaling aerosols of either saline (S), ammonium sulfate (AS), or sulfuric acid (SA). Two inhalations of 10 min were performed 30 min apart. Thirty minutes later, a hyperventilation challenge with 0.75 ppm SO_2 was performed. We determined the ventilation rate necessary to increase SR_{aw} by 100%, $PV_{100}SR_{aw}$, by stepwise increasing ventilation. The response showed large variation after AS and SA, with no significant differences between the first and the second inhalation. SR_{aw} was significantly increased after AS. Mean $PV_{100}SR_{aw}$ did not differ between S, AS, and SA. In a second experiment we investigated in 6 patients with bronchial asthma the effect of short-term inhalation of ammonium nitrate aerosol. We did not observe an effect of nitrate aerosol of airway responsiveness to sulfur dioxide. From these data we conclude that in patients with mild asthma short-term inhalation of sulfate aerosols produces small effects on lung function, no refractoriness, and no change of bronchial responsiveness to hyperventilation of SO_2. In addition, short-term inhalation of ammonium nitrate aerosol does not affect bronchial responsiveness to sulfur dioxide.

Relationship between Urinary Cotinine Level and Diagnosis in Children Admitted to Hospital—AC Reese, IR James, LI Landau, PN Lesouëf. Am Rev Respir Dis 1992;146:66.

The reported association between passive smoking and respiratory illness in children has been based on the parents' assessment of their own level of smoking. To more critically evaluate a causal relationship between passive smoking and childhood ill health, we used urinary cotinine, which is the major metabolite of nicotine and has a long half-life, to objectively quantitate the level of passive smoking in children. Urine was collected from 609 children (median age 3.8 y, range 1 month to 17 y) on admission to hospital; cotinine levels were obtained in 491 of these samples, and a comprehensive respiratory questionnaire was completed for 468 children. Statistical analysis was carried out on transformed data using both parametric and nonparametric statistics. Cotinine levels in the children correlated with the parents' current smoking ($p < 0.001$). Elevated levels were found in the 41 children admitted with bronchiolitis compared with a group of similarly aged children with nonrespiratory illnesses ($p < 0.02$). Elevated levels were not found for any other diagnosis. We conclude that the urinary cotinine approach has provided objective evidence linking passive smoking to hospital admission for bronchiolitis in infants.

Recommendations for Improving Cardiopulmonary Resuscitation Skills Retention—DK Moser, S Coleman. Heart Lung 1992;21:372.

Massive community efforts are devoted to delivering cardiopulmonary resuscitation (CPR) training to health professionals and lay people. However, although most people can successfully learn to perform CPR, skills retention is universally poor. Beginning as early as 2 weeks after initial training, CPR skills begin to deteriorate in a wide variety of subjects including nurses, physicians, emergency medical technicians, family members of patients with cardiac disease, and other lay people. Methods tested to improve retention are reviewed, and the role of practice and review is examined. The failure of many factors to improve retention of CPR skills is discussed. Finally, suggestions for improvement in retention of CPR skills based on a review of the literature and pertinent theory are offered.

Changes in Crackle Characteristics during the Clinical Course of Pneumonia—P Piirilä. Chest 1992; 102:176.

Recorded crackling lung sounds of 11 patients with pneumonia were studied with phonopneumography, FFT spectrography and time-expanded waveform display. The sounds were recorded on average 6 days after the onset of pneumonia and the recording was repeated 2 to 4 days later. In the first recording the crackles were coarse and midinspiratory. The patients with unilateral pneumonia had a significant difference in the upper frequency limit of inspiratory sound of the FFT spectrum between the healthy and diseased lung ($p < 0.01$). In the second recording, the beginning of crackling had shifted later ($p < 0.01$) and the end point of crackling also became later ($p < 0.05$). The largest deflection width of the individual crackles became shorter ($p < 0.05$). The results indicate that the pneumonic crackles vary markedly during the clinical course of pneumonia. The duration of the individual crackles became shorter and the timing of the crackles shifted toward the end of inspiration.

Trust, but Verify: The Accuracy of References in Four Anesthesia Journals—MF McLellan, LD Case, MC Barnett. Anesthesiology 1992; 77:185.

To determine the accuracy of bibliographic citation in the anesthesia literature, we reviewed all 1988 vol-

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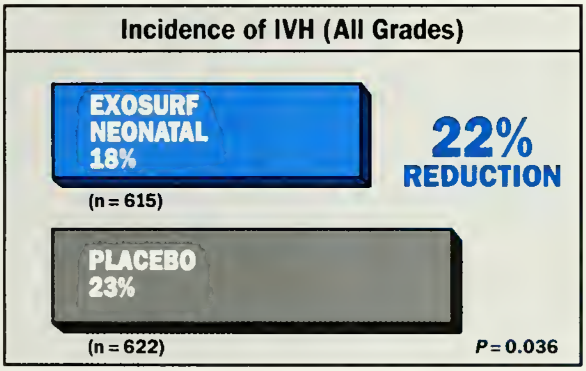

Two-dose treatment. Incidence of IVH in infants weighing 1250 g or more.
Adapted from Long et al[2(p1700)]

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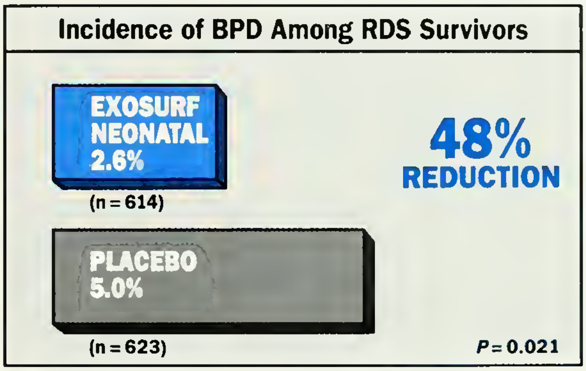

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Adapted from Long et al[2(p1699)]

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INDICATIONS AND USAGE: Exosurf Neonatal is indicated for: 1 **Prophylactic** treatment of infants with birth weights of less than 1350 grams who are at risk of developing RDS (see PRECAUTIONS), 2. **Prophylactic** treatment of infants with birth weights greater than 1350 grams who have evidence of pulmonary immaturity, and 3 **Rescue** treatment of infants who have developed RDS.

CONTRAINDICATIONS: There are no known contraindications to treatment with Exosurf Neonatal

WARNINGS: Intratracheal Administration Only: Exosurf Neonatal should be administered only by instillation into the trachea (see DOSAGE AND ADMINISTRATION) **General:** The use of Exosurf Neonatal requires expert clinical care by experienced neonatologists and other clinicians who are accomplished at neonatal intubation and ventilatory management. Adequate personnel, facilities, equipment, and medications are required to optimize perinatal outcome in premature infants Vigilant clinical attention should be given to all infants prior to, during, and after administration of Exosurf Neonatal **Acute Effects:** Exosurf Neonatal can rapidly affect oxygenation and lung compliance **Lung Compliance:** If chest expansion improves substantially after dosing, peak ventilator inspiratory pressures should be reduced immediately, without waiting for confirmation of respiratory improvement by blood gas assessment Failure to reduce inspiratory ventilator pressures rapidly in such instances can result in lung overdistention and fatal pulmonary air leak **Hyperoxia:** If the infant becomes pink and transcutaneous oxygen saturation is in excess of 95%, FiO_2 should be reduced in small but repeated steps (until saturation is 90 to 95%) without waiting for confirmation of elevated arterial pO_2 by blood gas assessment Failure to reduce FiO_2 in such instances can result in hyperoxia **Hypocarbia:** If arterial or transcutaneous CO_2 measurements are <30 torr, the ventilator rate should be reduced at once. Failure to reduce ventilator rates in such instances can result in marked hypocarbia, which is known to reduce brain blood flow **Pulmonary Hemorrhage:** In the single study conducted in infants weighing <700 grams at birth, the incidence of pulmonary hemorrhage (10% vs 2% in the placebo group) was significantly increased in the Exosurf Neonatal group. None of the five studies involving infants with birth weights >700 grams showed a significant increase in pulmonary hemorrhage in the Exosurf Neonatal group In a cross-study analysis of these five studies, fatal pulmonary hemorrhage occurred in three infants, two in the Exosurf Neonatal group and one in the placebo group Mortality from all causes among infants who developed pulmonary hemorrhage was 43% in the placebo group and 37% in the Exosurf Neonatal group Pulmonary hemorrhage in both Exosurf Neonatal and placebo infants was more frequent in infants who were younger, smaller, male, or who had a patent ductus arteriosus Pulmonary hemorrhage typically occurred in the first 2 days of life in both treatment groups. **Mucous Plugs:** Infants whose ventilation becomes markedly impaired during or shortly after dosing may have mucous plugging of the endotracheal tube, particularly if pulmonary secretions were prominent prior to drug administration Suctioning of all infants prior to dosing may lessen the chance of mucous plugs obstructing the endotracheal tube If endotracheal tube obstruction from such plugs is suspected, and suctioning is unsuccessful in removing the obstruction, the blocked endotracheal tube should be replaced immediately

PRECAUTIONS: General: In the controlled clinical studies, infants known prenatally or postnatally to have major congenital anomalies, or who were suspected of having congenital infection, were excluded from entry However, these disorders cannot be recognized early in life in all cases, and a few infants with these conditions were entered The benefits of Exosurf Neonatal in the affected infants who received drug appeared to be similar to the benefits observed in infants without anomalies or occult infection **Prophylactic Treatment—Infants <700 Grams:** In infants weighing 500 to 700 grams, a single prophylactic dose of Exosurf Neonatal significantly improved FiO_2 and ventilator settings, reduced pneumothorax, and reduced death from RDS, but increased pulmonary hemorrhage (see WARNINGS) Overall mortality did not differ significantly between the placebo and Exosurf Neonatal groups Data on multiple doses in infants in this weight class are not yet available **Rescue Treatment—Number of Doses:** A small number of infants with RDS have received more than two doses of Exosurf Neonatal as rescue treatment Definitive data on the safety and efficacy of these additional doses are not available **Carcinogenesis, Mutagenesis, Impairment of Fertility:** Exosurf Neonatal at concentrations up to 10,000 µg/plate was not mutagenic in the Ames Salmonella assay Long-term studies have not been performed in animals to evaluate the carcinogenic potential of Exosurf Neonatal The effects of Exosurf Neonatal on fertility have not been studied

ADVERSE REACTIONS:
General: Premature birth is associated with a high incidence of morbidity and mortality Despite significant reductions in overall mortality associated with Exosurf Neonatal, some infants who received Exosurf Neonatal developed severe complications and either survived with permanent handicaps or died In controlled clinical studies evaluating the safety and efficacy of Exosurf Neonatal, numerous safety assessments were made In infants receiving Exosurf Neonatal, pulmonary hemorrhage, apnea and use of methylxanthines were increased A number of other adverse events were significantly reduced in the Exosurf Neonatal group, particularly various forms of pulmonary air leak and use of pancuronium. ***Reflux:*** Reflux of Exosurf Neonatal into the endotracheal tube during dosing has been observed and may be associated with rapid drug administration. If reflux occurs, drug administration should be halted and, if necessary, peak inspiratory pressure on the ventilator should be increased by 4 to 5 cm H_2O until the endotracheal tube clears. ***>20% Drop in Transcutaneous Oxygen Saturation:*** If transcutaneous oxygen saturation declines during dosing, drug administration should be halted and, if necessary, peak inspiratory pressure on the ventilator should be increased by 4 to 5 cm H_2O for 1 to 2 minutes In addition, increases of FiO_2 may be required for 1 to 2 minutes.

DOSAGE AND ADMINISTRATION: Preparation of Suspension: Exosurf Neonatal is best reconstituted immediately before use because it does not contain antibacterial preservatives. However, the reconstituted suspension is chemically and physically stable when stored at 2° to 30°C (36° to 86°F) for up to 12 hours following reconstitution Solutions containing buffers or preservatives should not be used for reconstitution. **Do Not Use Bacteriostatic Water for Injection, USP.** Each vial of Exosurf Neonatal should be reconstituted only with **8 mL** of the accompanying diluent (preservative-free Sterile Water for Injection). **Dosage: Accurate determination of weight at birth is the key to accurate dosing.** ***Prophylactic Treatment:*** The first dose of Exosurf Neonatal should be administered as a single 5 mL/kg dose as soon as possible after birth Second and third doses should be administered approximately 12 and 24 hours later to all infants who remain on mechanical ventilation at those times. ***Rescue Treatment:*** Exosurf Neonatal should be administered in two 5 mL/kg doses The initial dose should be administered as soon as possible after the diagnosis of RDS is confirmed. The second dose should be administered approximately 12 hours following the first dose, provided the infant remains on mechanical ventilation **Use of Special Endotracheal Tube Adapter:** With each vial of Exosurf Neonatal for Intratracheal Suspension, five different sized endotracheal tube adapters each with a special right angle Luer®-lock sideport are supplied The adapters are clean but not sterile **Administration:** The infant should be suctioned prior to administration of Exosurf Neonatal Exosurf Neonatal suspension is administered via the sideport on the special endotracheal tube adapter **WITHOUT INTERRUPTING MECHANICAL VENTILATION.** Each Exosurf Neonatal dose is administered in two 2 5 mL/kg half-doses Each half-dose is instilled slowly over 1 to 2 minutes (30 to 50 mechanical breaths) in small bursts timed with inspiration. After the first 2.5 mL/kg half-dose is administered in the midline position, the infant's head and torso are turned 45° to the **right** for 30 seconds while mechanical ventilation is continued After the infant is returned to the midline position, the second 2.5 mL/kg half-dose is given in an identical fashion over another 1 to 2 minutes. The infant's head and torso are then turned 45° to the **left** for 30 seconds while mechanical ventilation is continued, and the infant is then turned back to the midline position These maneuvers allow gravity to assist in the distribution of Exosurf Neonatal in the lungs During dosing, heart rate, color, chest expansion, facial expressions, the oximeter, and the endotracheal tube patency and position should be monitored **Suctioning should not be performed for two hours after Exosurf Neonatal is administered, except when dictated by clinical necessity**

HOW SUPPLIED: Exosurf Neonatal for Intratracheal Suspension is supplied in a carton containing one 10 mL vial of Exosurf Neonatal for Intratracheal Suspension, one 10 mL vial of Sterile Water for Injection, and five endotracheal tube adapters (2 5, 3.0, 3.5, 4 0, and 4 5 mm I.D.). (NDC 0081-0207-01). Store Exosurf Neonatal for Intratracheal Suspension at 15° to 30°C (59° to 86°F) in a dry place

EDUCATIONAL MATERIAL: A videotape on dosing is available from your Burroughs Wellcome Co. representative. This videotape demonstrates techniques for safe administration of Exosurf Neonatal and should be viewed by health care professionals who will administer the drug

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Measurement of Alveolar Gas Mixing in Mechanically Ventilated Patients—WJ Kox, CJ Mills. Crit Care Med 1992;20:924.

OBJECTIVE: To evaluate a computer-based, realtime, multibreath nitrogen washout technique in mechanically ventilated patients, incorporating an in-line flow measurement device to measure functional residual capacity and two indices of gas mixing, ventilatory efficiency, and alveolar mixing efficiency. SETTING: ICU, Charing Cross Hospital, London. DESIGN: Within-patient reproducibility of a multibreath nitrogen washout technique. PATIENTS: 7 intubated patients requiring mechanical ventilation. 1 patient completed two sets of readings. INTERVENTIONS: Patients were connected to a pneumatically driven ventilator fitted with a switching device to be operated either by an appropriate oxygen-nitrogen mixture or equivalently blended oxygen-argon mixture. An inspiratory-expiratory, two-way valve was attached to the delivery port of the ventilator, with a pneumotachograph for flow measurement and a gas sampling probe for gas concentration measurement in line with the patient's endotracheal tube. The analog signals were digitized and handled by a microcomputer. MEASUREMENTS & MAIN RESULTS: No significant differences were found for any index, with coefficients of variation of 1.5%, 2.9%, and 2.1% for functional residual capacity, ventilatory efficiency, and alveolar mixing efficiency, respectively. CONCLUSIONS: This method gives excellent reproducibility for biological measurements in a clinical setting and shows that these measurements can readily be made on mechanically ventilated patients.

Metabolic Requirements in Tetanus—DM Linton, Y Wells, PD Potgieter. Crit Care Med 1992;20:950.

OBJECTIVE: To measure the metabolic requirements of patients with severe tetanus who require mechanical ventilation. DESIGN: Prospective, consecutive, open study using routine monitoring. SETTING: A multidisciplinary ICU in a large teaching hospital. PATIENTS: 5 consecutive patients (age range 30-54 yrs) with severe tetanus. 4 patients had clinical evidence of sympathetic nervous system overactivity. INTERVENTIONS: All patients were mechanically ventilated and appropriately treated for severe tetanus. Sympathetic nervous system overactivity was reduced by the administration of sedatives. MEASUREMENTS & MAIN RESULTS: Measurements of metabolic rates were made using an indirect calorimetry device. Each of the 5 patients had 3 8-hr periods of continuous metabolic monitoring for each of 3 levels of daily enteral nutritional support. The measured metabolic rates varied from 1,310 to 2,050 kcal/24 hrs (predicted 1,280 to 1,770 kcal/24 hrs). The variations from predicted basal metabolic rates varied from –6.3% to +10.5%. CONCLUSIONS: The measured metabolic rates of patients with severe tetanus who are appropriately sedated are relatively constant and are within 10.5% of the predicted basal metabolic rates.

Antioxidant Treatment with N-Acetylcysteine during Adult Respiratory Distress Syndrome: A Prospective, Randomized, Placebo-Controlled Study—S Jepsen, P Herlevsen, P Knudsen, MI Bud, N-O Klausen. Crit Care Med 1992;20:918.

OBJECTIVE: To examine whether the antioxidant N-acetylcysteine could ameliorate the course of the adult respiratory distress syndrome (ARDS) in man. DESIGN: Randomized, double-blind, placebo-controlled study. SETTING: Medical and surgical ICU in a regional hospital. PATIENTS: 66 ICU patients with ARDS. INTERVENTIONS: Patients with ARDS (P_{aO_2}/F_{IO_2} ratio < 250 torr) were treated with either the

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antioxidant N-acetylcysteine 150 mg/kg as a loading dose and then 20 mg/kg/hr, or with placebo for 6 days. MEASUREMENTS & MAIN RESULTS: No improvement could be demonstrated in the P_{aO_2}/F_{IO_2} ratio in the study group as compared with the control group on any day. Pulmonary compliance was higher in the N-acetylcysteine group than in the placebo group on all days, but this difference did not reach the chosen 5% level of significance. No difference between the two groups could be demonstrated on chest radiograph or on survival rate. We documented that N-acetylcysteine acts as an anticoagulant and perhaps decreases pulmonary fibrin uptake during ARDS. CONCLUSIONS: N-acetylcysteine might be of benefit in ARDS. Before further clinical studies are started, problems with N-acetylcysteine and coagulation have to be elucidated in order to find out whether N-acetylcysteine could have a beneficial effect in the treatment of ARDS.

Use of Arm Crank Exercise in the Detection of Abnormal Pulmonary Gas Exchange in Patients at Low Altitude—TW Martin, RJ Zeballos, IM Weisman. Chest 1992;102:169.

BACKGROUND: The measurement of arterial blood gases, $P_{(A-a)O_2}$ and V_D/V_T, during cycle ergometry is the "gold standard" for the assessment of pulmonary gas exchange. However, some patients are unable to perform cycle ergometry because of other medical problems. STUDY OBJECTIVE: To determine whether arm crank exercise could be used to reliably detect gas exchange abnormalities compared to cycle ergometry. PARTICIPANTS: 15 patients with a variety of pulmonary disorders, who were referred for exertional dyspnea. DESIGN: All patients performed maximal arm crank and cycle exercise. Arterial blood gases, $\dot{V}_{O_2}$, $\dot{V}_{CO_2}$, and $\dot{V}_E$ were measured at rest and during exercise. RESULTS: Compared to peak cycle exercise (mean ± SD), P_{aO_2} (85 ± 14 vs 75 ± 13 torr, S_{aO_2} (94 ± 2 vs 91±4 %), V_D/V_T (0.21 ± 0.07 vs 0.19 ± 0.08), and pH (7.37 ± 0.04 vs 7.34 ± 0.03) were significantly higher during peak arm crank exercise. The $P(A-a)O_2$ (18 ± 13 vs 29 ± 12 torr) was narrower, and P_{aCO_2} (29 ± 3 vs 29 ± 4 torr) and P_{AO_2} (104 ± 4 vs 103 ± 4 torr) were similar. Six patients had normal gas exchange during cycle exercise at low altitude $P_{(A-a)O_2}$ 27 torr, P_{aO_2} 65 torr, V_D/V_T 0.18) and nine were abnormal. Utilizing criteria specific for arm crank at low altitude, the same six patients had normal gas exchange $P_{(A-a)O_2}$ 13 torr, $P_{aO_2} > 85$ torr, V_D/V_T 0.26), and the remaining nine were abnormal. The $P_{(A-a)O_2}$ during peak arm crank was the most useful criteria in identifying patients with abnormal gas exchange. CONCLUSION: Proposed criteria for arm crank exercise testing accurately identified all patients with normal and abnormal pulmonary gas exchange during cycle exercise. The data from the present study suggest that arm crank can be an acceptable alternative exercise testing modality for the assessment of pulmonary gas exchange.

The Role of Bronchoscopy in the Diagnosis of Pulmonary Tuberculosis in Patients at Risk for HIV Infection—SH Salzman, ML Schindel, CP Aranda, RL Smith, ML Lewis. Chest 1992;102:143.

The present study was undertaken to clarify the role of bronchoalveolar lavage (BAL) and transbronchial bi-

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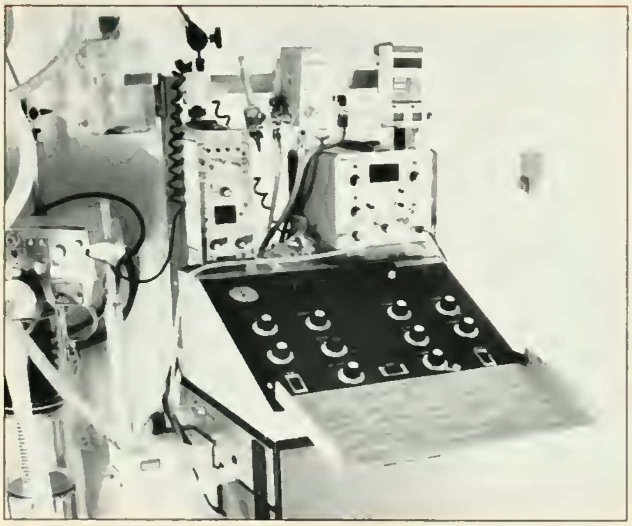

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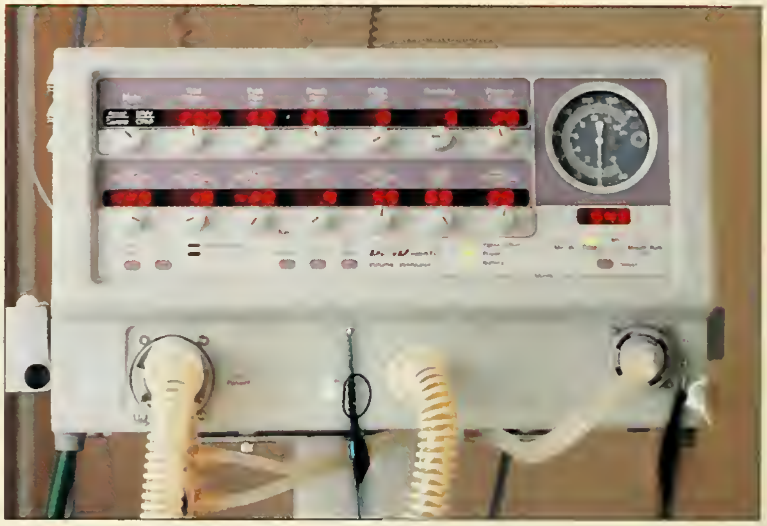

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opsy (TBB) in the diagnosis of pulmonary tuberculosis in patients at risk for human immunodeficiency virus (HIV) infection. We retrospectively identified 31 patients at risk for HIV who proved to have *Mycobacterium* tuberculosis on culture of at least one pulmonary specimen. All had pulmonary symptoms but initial sputum smears negative for acid-fast bacilli (AFB). All underwent fiberoptic bronchoscopy (FOB), including BAL and TBB; postbronchoscopy sputum was also collected in 19 patients. A specimen was considered to yield an immediate diagnosis when positive for AFB either on smear or histologic study; granulomas alone were considered positive when no other causes were identified. Overall, an immediate diagnosis was made by bronchoscopic specimens in 15 (48%) of 31 cases. TBB was the sole positive specimen in 7 patients (23 %). For comparison, similar specimens from 40 patients in whom *M avium complex* (MAC) grew on culture were also evaluated. An immediate identification of AFB was made in only 4 patients (10%). We conclude that the finding of AFB on staining of any pulmonary specimen is highly suggestive of tuberculosis, rather than MAC, and warrants institution of antituberculosis therapy. Of all bronchoscopic specimens, TBB provides the highest yield for an immediate diagnosis of tuberculosis.

Reliability of a Respiratory History Questionnaire and Effect of Mode of Administration on Classification of Asthma in Children—JK Peat, CM Salome, BG Toelle, A Bauman, AJ Woolcock. Chest 1992; 102:153.

Because there is no consensus definition of asthma for epidemiology, we have examined the reliability of a questionnaire and the effect of its mode of administration on classification of asthma in children. A symptom history questionnaire was parent self-administered and then re-administered within 3 months by a nurse. The questions of diagnosed asthma, cumulative wheeze, and recent wheeze (wheeze in the previous 12 mon) were more repeatable than questions of night cough, but 7% of children changed diagnosed asthma category, 13% changed cumulative wheeze category, and 9% changed recent wheeze category at second questionnaire. Because the numbers who changed from symptom positive to negative roughly equalled the changes from negative to positive, prevalence estimates were not affected. Methods of measuring asthma with greater precision are urgently needed. Because of reporting bias, epidemiologic information collected by current questionnaires should be treated with some caution.

Evaluation of Breath-by-Breath Measurement of Respiratory Gas Exchange in Pediatric Exercise Testing—T Reybrouck, F Deroost, LG Van Der Hauwaert. Chest 1992; 102:147.

In adults, breath-by-breath analysis has been used for measuring respiratory gas exchange during exercise. The present study evaluates the validity and reproducibility of this method in children. In 21 patients with various types of congenital heart disease, steady state exercise testing was performed on a motor-driven treadmill. Based on simultaneous measurements of $\dot{V}_{O_2}$, $\dot{V}_{CO_2}$, $\dot{V}_E$, and R, comparisons were made between the breath-by-breath and Douglas-bag methods. No significant differences were found between both methods for any of the variables. In 7 other patients the reproducibility of cardiorespiratory variables during exercise was assessed. No significant difference was found for the cardiorespiratory variables

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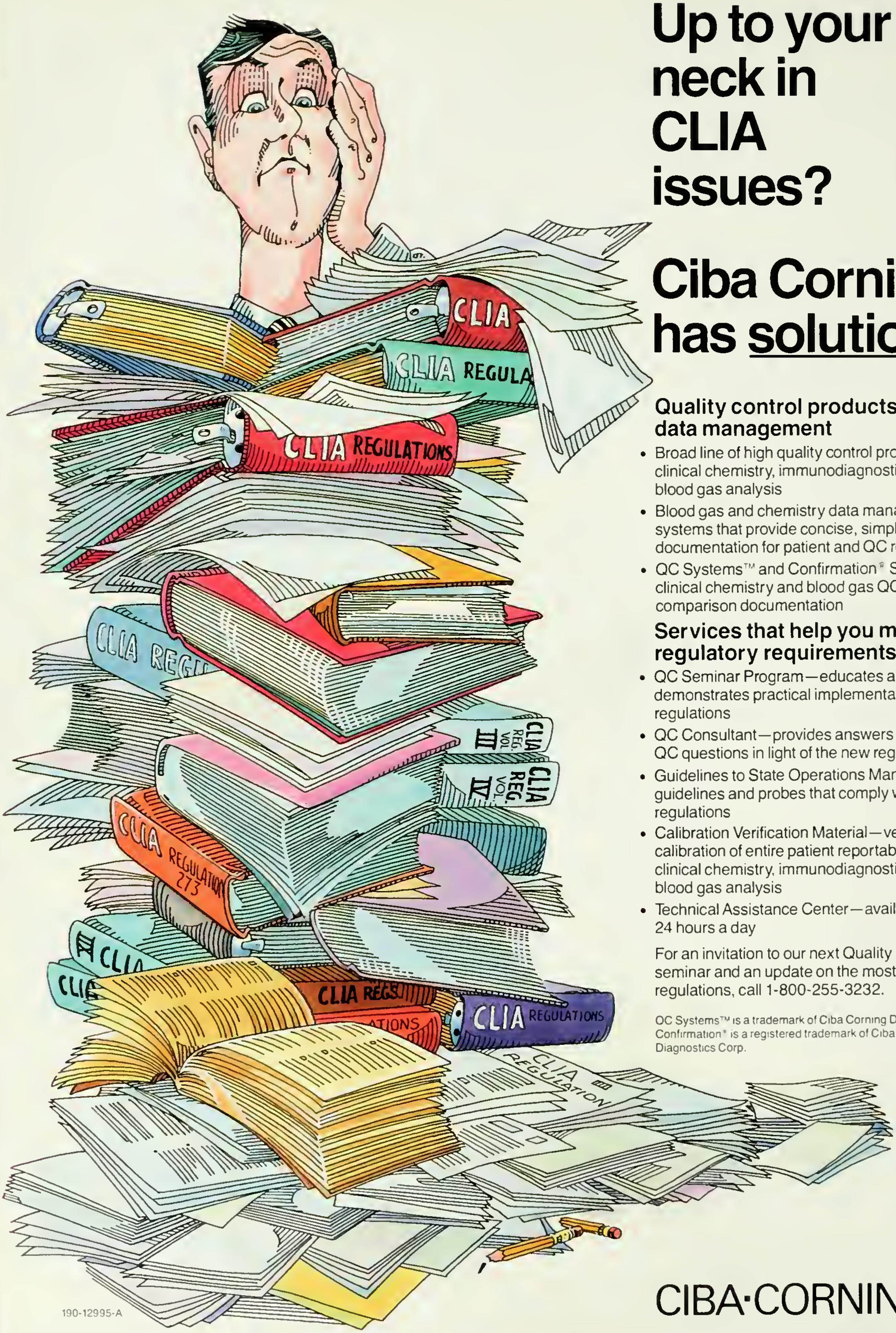

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during any of the tests and the coefficients of variation were comparable to data obtained in adults. It is concluded that the breath-by-breath method for measuring respiratory values can be applied in children with an acceptable degree of validity and reproducibility.

Evaluation of Respiratory Inductive Plethysmography in the Measurement of Breathing Pattern and PEEP-Induced Changes in Lung Volume—P Valta, J Takala, R Foster, C Weissman, JM Kinney. Chest 1992;102:234.

STUDY OBJECTIVE: To assess the accuracy of the respiratory inductive plethysmography in the measurement of PEEP-induced changes in end-expiratory lung volume during mechanical ventilation and its accuracy and stability in the measurement of ventilation during controlled mechanical ventilation and spontaneous breathing. DESIGN: An open comparison between two methods using a criterion standard. Either a pneumotachometer (mechanically ventilated patients) or a spirometer (spontaneously breathing subjects) was used as the reference method. SETTING: Tertiary care center; a multidisciplinary intensive care unit and a metabolic research unit. PATIENTS: 6 mechanically ventilated, paralyzed postoperative open heart surgery patients, 6 spontaneously breathing COPD patients, and 8 healthy volunteers. INTERVENTIONS: Stepwise increases and reductions of PEEP from 0 to 12 cm H_2O during controlled mechanical ventilation; repeated validation of the calibration of the respiratory inductive plethysmography (RIP) in both mechanically ventilated and spontaneously breathing subjects. MEASUREMENTS & RESULTS: The baseline drift of the RIP in vitro was 10 mL/150 min and in a ventilated model it was 20 mL/150 min. In mechanically ventilated patients, the mean error of the calibration after 150 min was within ±5%. Change in end-expiratory lung volume (EELV) during the stepwise increase of PEEP up to 12 cm H_2O was 849 ± 13 mL with the RIP and 809 ± 125 mL with the pneumotachometer (PT), and during the stepwise reduction of PEEP it was 845 ± 124 mL and 922 ± 122, respectively (not significant [NS]). The mean difference between methods in the measurement of change in EELV was –6.6 ± 3.5 % during increasing and 6.6 ± 6.7 % during decreasing PEEP (NS). Both in mechanically ventilated and spontaneously breathing subjects, the difference between methods was significant for V_T and V_T/t_i. The difference in V_T was –2.2 ± 0.2% during mechanical ventilation, –1.1 ± 0.5% in spontaneously breathing COPD patients, and 2.9 ± 0.4% in healthy volunteers (NS between groups). CONCLUSIONS: The RIP is sufficiently accurate for the measurement of PEEP-induced changes in EELV during controlled mechanical ventilation. The accuracy of tidal volume measurement is similar during mechanical ventilution and spontaneous breathing. The calibration of the RIP is stable enough for bedside monitoring of changes in lung volumes.

Survival of Patients with Severe Thoracic Spine Deformities Receiving Domiciliary Oxygen Therapy—K Ström, K Pehrsson, J Boe, A Nachemson. Chest 1992;102:164.

Scoliosis can lead to respiratory failure and premature death. Alveolar hypoventilation is a dominant cause and artificial ventilation at home (AVH) is probably the treatment of choice. It has been suggested that long-term domiciliary oxygen therapy (LTO) is of little value because of the worsening of hypercapnia. We analyzed survival and predictors of death among 80 patients with scoliosis and other severe thoracic spine deformities receiving LTO for chronic hypoxia. The survival rate was higher in patients under the age of 65 ($p = 0.01$) and in patients without concomitant pulmonary or airways disease. Likewise, the survival rate was higher in patients with a P_{aCO_2} of > 7.4 kPa than in patients with a lesser degree of hypoventilation and hypercapnia ($p < 0.05$). The risk of developing life-threatening hypercapnia during well-controlled LTO appeared to be small. In younger patients without complicating disease, long-term survival was achieved with LTO, but with time, an increasing proportion of the patients changed to AVH, with or without LTO.

A Two-Bag System for Continuous Measurement of Oxygen Uptake—JH Auchincloss, R Gilbert, GW Lighty Jr, D Peppi, CL Hare. Chest 1992;102:112.

Collection of mixed expired gas in a bag has been a classic method for the estimation of $\dot{V}_{O_2}$ during the steady state but has not been employed during unsteady state exercise in part because there is a need for suspending the acquisition of data during the period of gas analysis unless many bags are used. In this study a two-bag system is described in which one bag fills while the other is analyzed. Bag volume is under the control of the operator, and we employed volumes of 30-50 L. Thirty-one subjects were studied with this circuit in a progressive treadmill test. Although $\dot{V}_{O_2}$ could be falsely elevated during periods of overbreathing, this source of error could be identified and its effect reduced if $\dot{V}_{O_2}$ was plotted against both ventilation and power requirement. Plateau values of $\dot{V}_{O_2}$ were identified only in 6 subjects and the ventilatory threshold in 16.

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Nonphysician Transport of Intubated Pediatric Patients: A System Evaluation—AJ Beyer III, G Land, A Zaritsky. Crit Care Med 1992;20:961.

OBJECTIVE: To evaluate the occurrence of complications and patient deteriorations during the air and ground transportation of intubated pediatric patients, performed by a nonphysician-based team under the direction of an intensive care attending physician or fellow. DESIGN: Retrospective chart review. SETTING: A 600-bed university hospital with a 16-bed neonatal ICU and a 12-bed pediatric ICU. PATIENTS: All intubated pediatric patients (422 of 614 patients transported during the study period) transported by the dedicated neonatal/pediatric transport team from April 1988 to April 1990. MEASUREMENTS & MAIN RESULTS: The transport records of intubated pediatric patients were abstracted. Recorded data included age, weight, gestational age, vital signs, diagnosis, interventions received, and use of paralytic agents and sedatives/analgesics. Patients were analyzed in three groups: Group 1 (n = 295) included neonates; Group 2 (n = 66) included patients > 1 month and < 1 yr of age; and Group 3 patients (n = 61) were ages > 1 yr. Group 1 had 9 (3.1%) complications or patient deteriorations; 4 (1.4%) were related to the endotracheal tube. Group 2 had 1 (1.5%) airway complication and 1 deterioration. Group 3 had no complications or deteriorations. All but one of the airway complications were effectively handled by the transport team. At the referring hospital, the transport nurse or respiratory therapist intubated 62 (19.8%) patients in Group 1, 5 (7.5%) in Group 2, and 3 (4.9%) patients in Group 3. 67 (23%), 21 (32%), and 30 (49%) patients of Groups 1, 2, and 3, respectively, were paralyzed for transport. No complications were secondary to the use of paralytic agents or sedatives. CONCLUSIONS: Under proper medical guidance, well-trained nonphysician personnel can provide low-risk transport of intubated pediatric patients. Use of sedatives and paralytic drugs did not increase the risk of complications or patient deterioration.

Ethical Aspects of Pediatric Home Care—JD Lantos, AF Kohrman. Pediatrics 1992;89:920.

This essay is a discussion of ethical issues that arise in the provision of home health care to technology-dependent children. Different ethical norms, especially with regard to the degree of professional responsibility for outcomes, traditionally have applied to home care and hospital care. In particular, parents generally are expected to do their best, but are not expected to have the same specialized knowledge of risks and benefits with regard to particular interventions as health professionals. When home health care involves the use of advanced medical technology, it strains traditional conceptions of parental responsibilities to care for the health of their children at home. It can also strain traditional concepts of professional responsibilities to care for critically ill children in hospitals. We discuss some of the tensions that arise as medical, psychological, and economic forces lead to the increas-ing use of high technology in the care of children outside of traditional health care institutions.

The Effects of Respiratory Training with Inspiratory Flow-Resistive Loads in Premature Infants—S Tan, S Duara, GS Neto, M Afework, T Gerhardt, E Bancalari. Pediatr Res 1992;31:613.

Respiratory training of premature infants was performed to determine whether improved respiratory muscle strength and/or endurance would result. Twenty two premature infants were randomized into control and training groups for 2 wk, using inspiratory flow-resistive loads for training (75 cm $H_2O \cdot s \cdot L^{-1}$ in wk 1 and 90 cm $H_2O \cdot s \cdot L^{-1}$ in wk 2). Respiratory endurance was assessed by the time interval required for the development of a 5-torr rise in transcutaneous CO_2 tension during the hypoventilation induced by loaded breathing, using a moderately severe resistive load (250 cm $H_2O \cdot s \cdot L^{-1}$ at 1 $L \cdot min^{-1}$). Respiratory strength was assessed by the maximum negative airway pressure generated during occluded breaths, a pressure-time integral, and an effort index. Results revealed that respiratory muscle endurance, which was not initially different between control and trained groups, increased significantly after 2 wk in the trained group by 137% (median value, $p < 0.05$), whereas it remained unchanged in the control group (–24%). The trained group of infants also showed a significant decrease in baseline breathing frequency between the initial and final measurements taken 2 wk apart when compared with controls ($p < 0.05$) and a lesser increase in inspiratory time with loading in the final measurement as compared with the initial value ($p < 0.05$). There was no significant difference between the control and trained groups in initial or subsequent measures of respiratory muscle strength. Inspiratory flow-resistive load training appears to improve the respiratory endurance of premature infants in whom respiratory muscle fatigue has been described to play a role in the development of respiratory failure.

The Relationship between Rhythmic Swallowing and Breathing during Suckle Feeding in Term Neonates—O Bamford, V Taciak, JH Gewolb. Pediatr Res 1992;31:619.

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suckle feeding and breathing in human infants. To establish baseline data, we recorded breathing and swallowing activity during bottle feeds in 23 infants at 14-48 h postnatal age. Most swallows (overall mean 68%) were organized into runs, with intervals starting at 0.6-0.8 s and slowing to 1-1.3 s after 30-40 s. The proportion of run swallows to total swallows increased significantly with age. Swallow intervals were regular (coefficient of variation = 18-38%) compared with breathing (coefficient of variation = 50%). Both breathing rate and tidal volume were significantly reduced by the onset of suckle feeding, and the pattern of respiratory airflow became markedly irregular. Mild transient desaturation was common, but was not accompanied by changes in heart rate. Swallows could occur in all phases of breathing. Overall, equal numbers of swallows were preceded by expiration and inspiration, but twice as many were followed by expiration compared with inspiration. Swallows were classified by the respiratory phases both preceding and following the swallow. Swallows occurred in all possible classifications in each of the infants studied. The incidence of the most frequent classification (inspiration-swallow-expiration) was 24% overall (individual range 5-50%). The phase relation between swallows and breaths changed frequently but showed occasional short periods of stability during which the breathing became regular and tidal volume increased. We conclude that at < 48 h the normal infant has little coordination between swallowing and breathing rhythms and maintains rhythmic swallowing at the expense of eupnea.

Surfactant Lavage in a Piglet Model of Meconium Aspiration Syndrome—MS Paranka, WF Walsh, BB Stancombe. Pediatr Res 1992;31:625.

Meconium aspiration continues to be a major cause of morbidity and mortality in newborn infants, and is one of the most common indications for extracorporeal membrane oxygenation. Lab studies have suggested that meconium inactivates surfactant and displaces surfactant from the alveolar surface. A recent report has suggested a clinical role for surfactant therapy in human infants with meconium aspiration. We evaluated the effect of surfactant (Survanta) lavage on a piglet model of meconium aspiration. Meconium pneumonitis was created by administration of 4 mL/kg of a 20% slurry of human meconium via endotracheal tube. Twenty-four newborn piglets were then randomly assigned to one of three groups: (1) suction only (n = 7), (2) saline lavage (n = 5), or (3) surfactant lavage (n = 7). Five piglets were excluded from analysis due to death from pneumothorax during meconium administration (n = 3), death from pneumothorax during saline lavage (n = 1), and death from pneumo-thorax during surfactant lavage (n = 1). The surfactant group had a statis-tically significant ($p < 0.05$) improvement in arterial-to-alveolar-oxygen-ratio gradient versus both control groups for the first 3 h. The oxygenation index was statistically significant versus the suction only group at 1, 3, and 4 h. Surfactant lavage of meconium aspiration in piglets results in short-term improvement of oxygenation and warrants further study.

What Is the Legal 'Standard of Medical Care' When There Is No Standard Medical Care? A Survey of the Use of Home Apnea Monitoring by Neonatology Fellowship Training Programs in the United States—W Meadow, D Mendez, J Lantos, R Hipps; M Ostrowski. Pediatrics 1992;89:1083.

In treating a patient, a doctor is obliged to use the skill and care that is ordinarily used by reasonably well-qualified doctors in similar cases. In addition, the only way in which a juror may decide whether the defendant used the skill and care that the law required of him or her is from evidence presented by doctors called as expert witnesses (cf *Illinois Pattern Jury Instructions*). However, what should be done if expert opinions differ concerning the care that is "ordinarily used"? Home apnea monitoring (HAM) is prescribed at times for graduates of neonatal intensive care units despite the fact that indications for its use are not well established and efficacy is completely unknown. The authors attempted to determine standards for HAM as it is currently practiced in neonatology training programs. The primary teaching hospital for each of the 99 neonatology training programs in the United States was identified. Both the medical director (MD) and a neonatal intensive care unit nurse manager (RN) were asked about the use of HAM in their own nursery for four clinical vignettes. Each vignette depicted a 1,000-g birthweight infant, currently 7 weeks old and ready for discharge. In three vignettes, the infant had demonstrated no apnea, mild apnea (resolved by 2 weeks of age), or moderate apnea (requiring theophylline therapy at discharge) during the hospital course. In the fourth vignette, the infant had no apnea but was to be discharged home with supplemental oxygen. For 67 of 99 training programs, paired responses of RN managers and MD directors were obtained. For infants with no apnea or mild apnea, approximately 85% of RN/MD pairs agreed that HAM would not be used at their institution, 2% would use HAM, and 12% responded that they might use HAM depending on individual circumstances. In contrast, for the premature infant with moderate apnea, there was much less agreement. Sixteen percent of RN/MD

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pairs responded that HAM would not be used, 39% would use HAM, and 19% might. Remarkably, for this vignette 25% of the RN/MD responses disagreed on the practice of HAM at their own center. Similarly, for the infant with home oxygen, 15% of RN/MD responses agreed that HAM would not be used, 49% answered that HAM would be used, 10% were uncertain, and 25% disagreed on the use of HAM at their own center. It is concluded that (1) for premature infants with no or mild apnea, HAM is currently prescribed by a minority of fellowship-associated neonatology programs, and (2) no obvious consensus exists for HAM in the context of moderate apnea or home oxygen therapy. For many infants, there is no "standard care" for HAM in the neonatology community at this time. "Expert" opinions of the legal "standard of care" for HAM should reflect this fact.

Utility of an End-Tidal Carbon Dioxide Detector during Stabilization and Transport of Critically Ill Children—MS Bhende, AE Thompson, RA Orr. Pediatrics 1992;89: 1042.

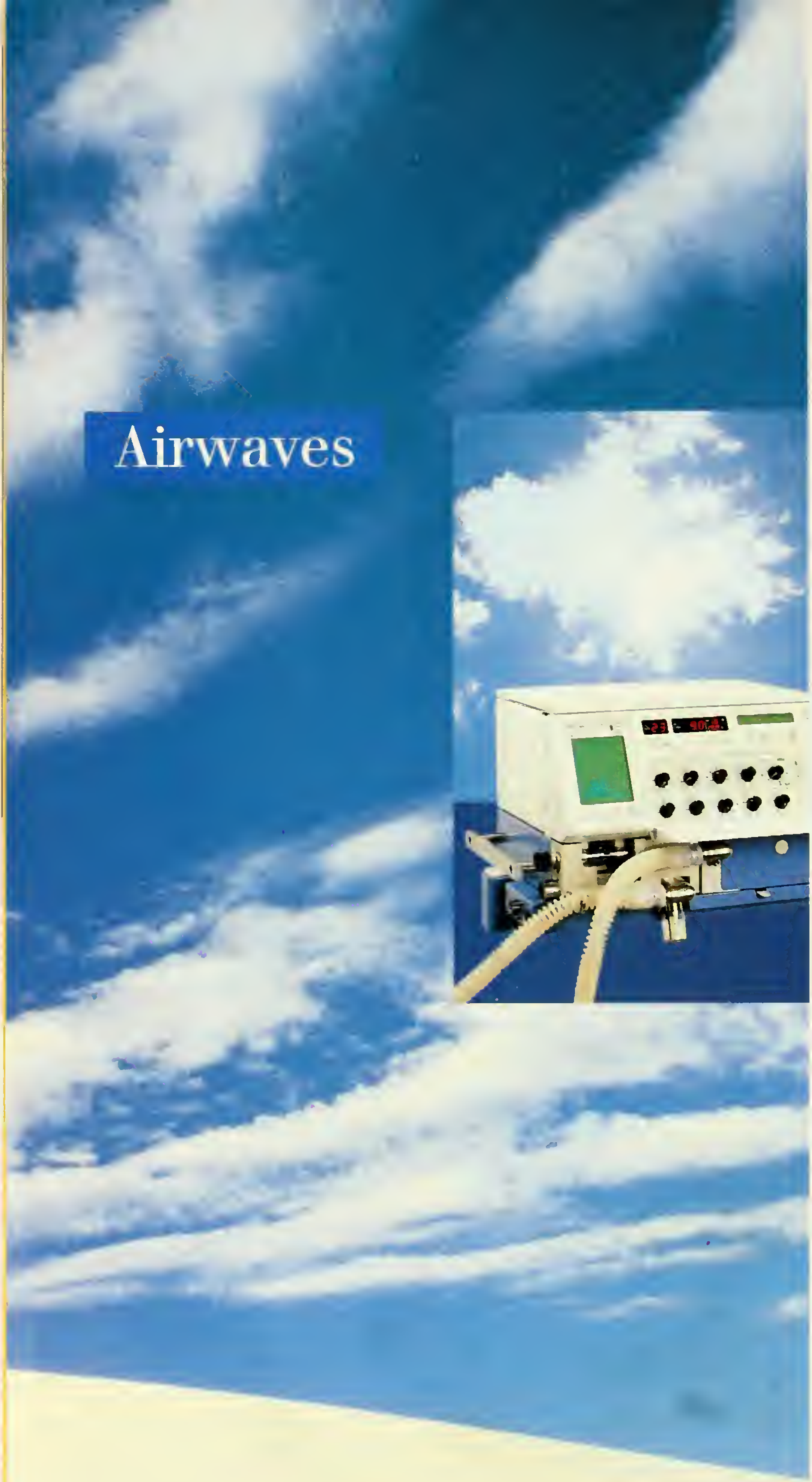
Critically ill children often require endotracheal intubation prior to transport to a medical center. Correct endotracheal tube placement and maintenance during transport are essential. The utility of a portable colorimetric end-tidal CO_2 detector during transport of critically ill children was evaluated. Fifty-eight children with spontaneous circulation (aged 1 day to 12 years, weight 0.9-26 kg) who underwent 59 intubations during transport by ground (n = 31) or air (n = 27) were studied. Tube position was confirmed by physical examination, arterial blood gas values, or arterial oxygen saturation, and sometimes by chest radiograph. The detector was attached and readings were obtained after intubation; readings were repeated if endotracheal tube position was rechecked during transport. Fifty-seven of 58 tracheal positions and the 1 esophageal tube position were correctly identified. One false-negative result occurred in a severely hypocarbic 900-g premature newborn. On each occasion that the detector was used en route, the endotracheal tube position was correctly identified. It is concluded that the end-tidal CO_2 detector is a useful tool for confirming endotracheal tube position during transport of critically ill children weighing more than 2 kg who are not in cardiopulmonary arrest.

Hypoxic Arousal Responses in Normal Infants—SLD Ward, DB Bautista, TG Keens. Pediatrics 1992; 89:860.

Failure to arouse in response to hypoxia has been described in infants at increased risk for sudden infant death syndrome (SIDS) and has been suggested as a possible mechanism for SIDS. However, most SIDS victims are not in a high-risk group before death. Thus, if a hypoxic arousal disorder is an important contributor to SIDS, normal infants might fail to arouse from sleep in response to hypoxia. To test this hypothesis, the authors studied hypoxic arousal responses in 18 healthy term infants younger than 7 months of age (age 12.1 ± 1.7 [SEM] weeks; 56% girls). Hypoxic arousal challenges were performed during quiet sleep by rapidly decreasing inspired oxygen tension (P_{IO_2}) to 80 torr for 3 minutes or until arousal (eye opening, agitation, and crying) occurred. Tests were performed in duplicate when possible. Only 8 infants (44%) aroused in response to one or more hypoxic challenges; arousal occurred during 8 (32%) of 25 trials. There were no significant differences in lowest P_{IO_2} or arterial oxygen saturation during hypoxia between those infants who aroused and those who failed to arouse. All 18 infants had a fall in their end-tidal carbon dioxide tension during hypoxia, suggesting that each had a hypoxic ventilatory response despite failure to arouse in the majority. Periodic breathing occurred following hypoxia in only 1 (13%) of the 8 trials that resulted in arousal compared with 16 (94%) of 17 trials without arousal ($p < 0.005$). It is concluded that the majority of normal infants younger than 7 months of age fail to arouse from quiet sleep in response to hypoxia, despite the apparent presence of a hypoxic ventilatory response.

Controlled Study of Linear Growth in Asthmatic Children during Treatment with Inhaled Glucocorticosteroids—OD Wolthers, S Pedersen. Pediatrics 1992;89: 839.

Linear growth was investigated with weekly knemometry in a population of 43 schoolchildren with mild asthma treated with inhaled budesonide. The study was a randomized, double-blind, parallel group study with three dose groups of 200, 400, and 800 μg of budesonide per day. Each dose group received budesonide for 8 consecutive weeks. Placebo was given for either 4 weeks before or after budesonide treatment. Twelve children in the 200-μg group, 14 in the 400-μg group, and 12 in the 800-μg group completed the 12-week study period. There was no significant difference in mean growth velocity among the three dose groups during placebo treatment. Compared with placebo (growth velocity: 0.39 mm/wk), mean lower leg growth velocity was reduced with 0.26 mm/wk ($p < 0.001$, T = 5.0, df = 11; 95% confidence interval 0.14-0.37 mm/wk) in children treated with 800 μg of budesonide. There was no statistically significant difference in growth velocity between 200- or 400-μg budesonide



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treatments and placebo. These data indicate that inhaled budesonide can be safely used in doses up to 400μg/d in schoolchildren with asthma.

Prospective Evidence of a Circadian Rhythm for Out-of-Hospital Cardiac Arrests—RL Levine, PE Pepe, RE Fromm Jr, PA Curka, PA Clark. JAMA 1992;267:2935.

OBJECTIVES: Published studies have indicated a circadian rhythm in the occurrence of sudden cardiac death. However, these studies have involved either retrospective analyses of death certificates or analyses of data collected during studies of pharmacologic agents in selected populations. PURPOSE: To determine whether a circadian pattern could be clearly demonstrated in a prospective study of out-of-hospital sudden cardiac death in a large, unselected population. DESIGN: All adult cases of sudden death of presumed primary cardiac cause from a large urban population were prospectively evaluated over a 12-mo period. The incidence of sudden cardiac death was analyzed using harmonic regression of the data tabulated by hour of the day. RESULTS: During the year of study, 1,019 consecutive primary cardiac arrests were analyzed. A significant circadian pattern was found ($p < 0.0001$) with the frequency of cardiac arrests increasing dramatically from 6 am until noon. CONCLUSIONS. This prospective study of out-of-hospital cardiac arrest confirms the existence of a circadian rhythm. These data have important implications for future investigation concerning the pathophysiology of sudden cardiac deaths.

Clinical Markers of Asthma Severity and Risk: Importance of Subjective As Well As Objective Factors—S Janson-Bjerklie, S Ferke-tich, P Benner, G Becker. Heart Lung 1992;21:265.

The purpose of this study was to explore patient perceptions of asthma severity and danger from asthma, correlate them with objective measures, and assess the impact of psychological variables on the perception of severity. Recognition of patients at greatest risk for fatal attacks requires identifying those with severe asthma. In our study of 95 adults with asthma, we found that the subjective factors of perceived severity and perceived danger and the objective factors of medications, hospitalizations, history of intubation, and pulmonary function were important markers of asthma severity and risk. Our findings indicate that asthmatic adults make independent self-assessments that generally correlate with objective markers of increased risk of mortality and increased severity of the asthma. The perception of high severity was significantly correlated with depression, panic-fear, frequency of emergency department visits, and with an objective index of risk of death. The latter includes variables obtainable from history alone (number of medications to control symptoms, need for prednisone, prior intubation, and prior recent hospitalization) and is correlated with spirometric indexes of airflow obstruction, occurrence of nocturnal symptoms, and number of emergency department visits.

Gastric Colonization and Pneumonia in Intubated Critically Ill Patients Receiving Stress Ulcer Prophylaxis: A Randomized, Controlled Trial—NM Apte, DR Karnad, TP Medhekar, GH Tilve, S Morye, GG Bhave. Crit Care Med 1992; 20:590.

OBJECTIVE: To study the effects of pharmacologically increasing gastric pH on gastric colonization and the development of pneumonia in intubated critically ill patients. DESIGN: Randomized, controlled trial. SETTING: Medical ICU in a university hospital. PATIENTS: 34 tracheotomized patients with tetanus. INTERVENTIONS: 16 patients received I.V. ranitidine to increase gastric pH > 4 (ranitidine group), while 18 patients received no prophylaxis for upper gastrointestinal bleeding (control group). MEASUREMENT & MAIN RESULTS: Mean gastric pH was higher in the ranitidine group (median 4.7, range 3.6-6.1) than in the control group (median 2.1, range 1.2-4.9; $p < 0.05$). Gastric colonization occurred in 15 (94%) of 16 patients who received ranitidine, 2 days (median; range 1-5) after intubation; gastric colonization also occurred in all control patients (median 4 days, range 1-9; $p < 0.05$). Pneumonia occurred in 13 (81%) of 16 patients who received ranitidine, 3 days (median, range 1-5) after intubation and in 9 (50%) of 18 control patients ($p < 0.01$) 5 days after tracheal intubation (median, range 3-14; $p < 0.01$). Prior gastric colonization by the pathogen that caused pneumonia was demonstrable in 9 (56%) of 16 patients who received ranitidine vs 8 (44%) of 18 control patients ($p > 0.05$). The risk for developing pneumonia in the ranitidine-treated group was highest in the first 4 days after tracheal intubation. There was no difference in the frequency of upper gastrointestinal hemorrhage in the two groups. CONCLUSIONS: Pharmacologically increasing gastric pH increases the risk for developing pneumonia in intubated critically ill patients. The pneumonia occurs earlier than in untreated control patients.

Cerebral Blood Flow Velocity in Normal, Full-Term Newborns Is Not Related to Ductal Closure—DG Batton, S Riordan, T Riggs. AJDC 1992;146:737.

OBJECTIVE: To correlate changes in blood flow velocity in the anterior

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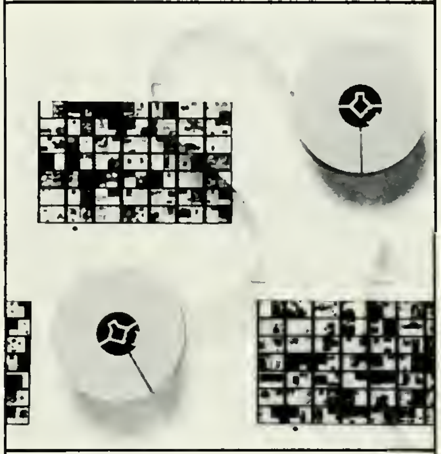
and middle cerebral arteries with closure of the ductus arteriosus in normal, full-term newborns during the first 2 days following delivery. DESIGN: Survey. SETTING: Large community hospital. PARTICIPANTS: 23 normal, full-term neonates. SELECTION PROCEDURES: Volunteer sample. INTERVENTIONS: None. MEASUREMENTS & RESULTS: We measured blood flow velocity in the anterior and middle cerebral arteries, cardiac output, and patency of the ductus arteriosus using pulsed Doppler, M-mode, and real-time ultrasound. The initial examination was performed at (mean ± SD) 7.6 ± 2 hours, and the second examination was performed at 30 ± 3 hours. The systolic, diastolic, and mean blood flow velocity in the anterior and middle cerebral arteries increased significantly from Day 1 to Day 2. Cardiac output did not change significantly (252 ± 49 vs 279 ± 69 $cm^3/kg/min$). Thirteen newborns on Day 1, but only two newborns on Day 2, had echocardiographic evidence of a patent ductus arteriosus. Newborns whose ductus was already closed on Day 1 had similar increases in blood flow velocity in the anterior and middle cerebral arteries from Day 1 to Day 2 compared with newborns whose ductus had closed from Day 1 to Day 2. CONCLUSION: The normal increase in blood flow velocity in the anterior and middle cerebral arteries in the first 2 days following delivery is not related to changes in cardiac output or ductal closure.

Process of Foregoing Life-Sustaining Treatment in a University Hospital: An Empirical Study—K Faber-Langendoen, DM Bartels. Crit Care Med 1992;20:570.

OBJECTIVES: The difficult decision to forego (withhold or withdraw) life-sustaining treatment has received extensive commentary. Little attention has been paid to how physicians do, and should, care for dying patients once this decision is made. This study describes the characteristics of patients who forego treatment, determines the range and sequential process of foregoing treatment, and suggests ethical and public policy implications. DESIGN: The charts of all patients who died at the University of Minnesota Hospital during a 2-mo period were reviewed. The patient information that was collected included age and sex, diagnoses, mental status, location in the hospital, length of hospital stay, method of payment, the timing of the first decision to forego treatment, and the range and sequence of interventions foregone. SETTING: All ICUs and general wards in a 586-bed tertiary care university hospital. PATIENTS: All patients who died at the University of Minnesota Hospital during May and June 1989. MAIN RESULTS: A total of 52 (74%) of 70 patients who died had some intervention withheld or withdrawn before death. Those patients in whom treatment was foregone were more likely to have an underlying malignancy or impaired mental status and longer hospital stays. 32 (62%) of 52 patients who declined some treatment were in an ICU; 26 (50%) of 52 patients required mechanical ventilation. On average, 5.4 separate interventions were foregone per patient. Resuscitation and/or endotracheal intubation were generally the first measures withheld; once a patient required a ventilator, withdrawing the ventilator was a late decision. Precise methods of ventilatory and vasopressor withdrawal varied considerably among patients. CONCLUSIONS: Foregoing life-sustaining treatment is not a single decision, but it often occurs in a sequential manner over several days. A strict analysis of the benefits and burdens of various interventions may be inadequate in deciding what interventions are appropriate in the care of the dying patient.



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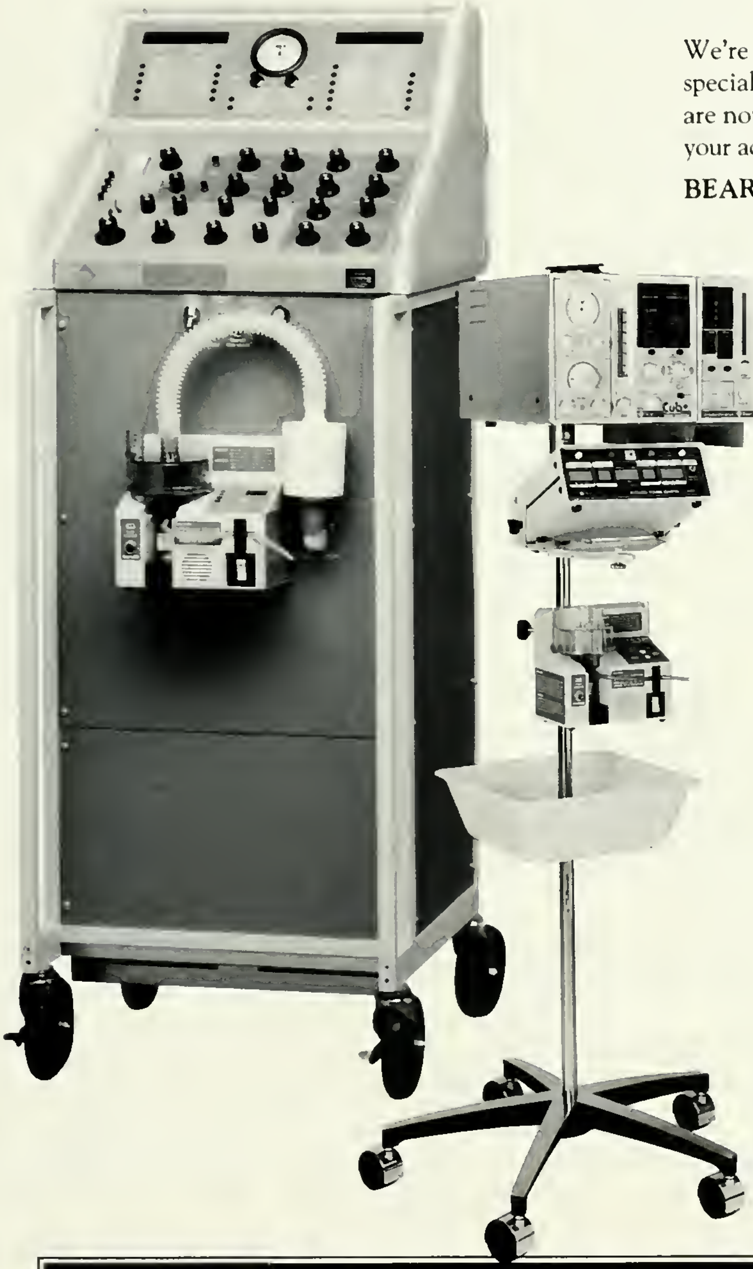

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CONSENSUS CONFERENCE ON THE ESSENTIALS OF MECHANICAL VENTILATORS

A Special Issue

containing the papers and consensus statement
comprising a conference
held February 27-29, 1992, in Cancun, Mexico,
sponsored by the American Association for Respiratory Care
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the American Respiratory Care Foundation

Faculty:

Richard D Branson RRT—Cincinnati, Ohio
Robert L Chatburn RRT—Cleveland, Ohio
Thomas D East PhD—Salt Lake City, Utah
Robert M Kacmarek PhD RRT—Boston, Massachusetts
Neil R MacIntyre MD—Durham, North Carolina
John J Marini MD—St Paul, Minnesota
David J Pierson MD—Seattle, Washington
Catherine Sassoon MD—Long Beach, California
Maire P Shelly MB ChB FFARCS—Manchester, United Kingdom
Martin J Tobin MD—Hines, Illinois

Invited Respondents:

David Burns MD—San Diego, California
T James Gallagher MD—Gainesville, Florida
John Overman BSEE—Kansas City, Kansas
Mark H Sanders MD—Pittsburgh, Pennsylvania
Arthur S Slutsky MD—Toronto, Ontario, Canada

Planning-Writing Committee:

Richard D Branson RRT—Cincinnati, Ohio
Pat Brougher RRT—Dallas, Texas
Robert L Chatburn RRT—Cleveland, Ohio
Thomas D East PhD—Salt Lake City, Utah
John J Marini MD—St Paul, Minnesota
Neil R MacIntyre MD—Durham, North Carolina

American Association for Respiratory Care

Consensus Statement on the Essentials of Mechanical Ventilators—1992

Introduction

The Consensus Conference on the Essentials of Mechanical Ventilators was convened by the American Association for Respiratory Care, the American Respiratory Care Foundation, and RESPIRATORY CARE Journal to identify the essential features of devices that provide mechanical ventilatory support through the application of positive airway pressure. The specific tasks were

1. to provide a standard nomenclature,
2. to identify the essential support features,
3. to identify essential heat and humidity requirements,
4. to identify essential monitoring/alarm features,
5. to identify what constitutes an order for mechanical ventilation.

It is important to note that the purpose of this conference was only to describe the essential features of these devices. The conference did not attempt to address the advantages or disadvantages of various strategies that use these features to apply positive pressure ventilatory support (eg, pressure vs volume breaths for total support, permissive hypercapnia vs inverse ratio ventilation to reduce airway pressures, IMV vs PSV for weaning). The purpose of this resulting document is to provide clinicians with the information necessary to make informed decisions regarding the purchase and operation of mechanical ventilators and to assist manufacturers in the development of devices that best serve the patient and clinician. This document is not intended to represent a standard of care or a manufacturing standard.

Certain terms describe the role of specific devices, components, or characteristics:

essential—considered to be necessary for safe and effective operation in the majority of patients in the specified setting

recommended—considered to be necessary for optimal management of virtually all patients in the specified setting

optional—considered to be possibly useful in limited situations but not necessary for a majority of patients in the specified setting

Classification and Terminology

The description of the features of a mechanical ventilator requires a standard nomenclature. These features can be classified using a scheme that focuses on key attributes or characteristics in a logical, consistent fashion. It is recommended that the following classification system be applied to mechanical ventilators and that the ventilator-performance-testing protocols specified in the American Society for Testing and Materials ASTM Document PF 1100-90 (Standard Specification for Ventilators Intended for Use in Critical Care) be utilized.[1]

Input Power

Input power is either pneumatic or electric.

Power Conversion and Transmission

Power conversion and transmission are accomplished by the ventilator's drive mechanism or mechanisms, which may incorporate either an external or internal compressor and the device's output control systems.

Control Scheme

The ventilator's *control circuit* is the subsystem responsible for controlling the drive mechanism (compressor and/or output control valves). This subsystem controls the *phase variables* in accordance with *conditional variables*.

A phase variable is a physical quality (pressure, volume, flow, or time) that is adjusted, measured, and/or used to manipulate some phase of the ventilatory cycle. These variables are controlled either by the ventilator or by the patient (ie, the patient's ventilatory drive and musculature functioning alone or in concert with interactive ventilator capabilities). When controlled by the ventilator, the phase variable remains constant despite changes in patient activity or changes in resistance and compliance. A ventilator-controlled variable is thus a variable that is considered to be the independent variable in the equation of motion for that phase of the breath:

$$pressure = \frac{volume}{compliance} + (flow)(resistance),$$

where pressure, volume, and flow are variables (functions of time), and compliance and resistance are constants. If pressure, volume, and flow all change as resistance and compliance change, then the ventilator-controlled variable is time.

The following terms apply to the inspiratory phase:

The trigger variable begins the phase.
The limit variable is a target value for pressure, volume, or flow that cannot be exceeded.
The cycle variable ends the phase.

Depending upon whether the ventilator or the patient controls triggering and cycling and upon the distribution of ventilatory work of the breath, four clinically different breath types (Table 1) can be classified under two broad engineering headings—machine-cycled and patient-cycled breaths.

Table 1. Breath Types Defined by Specific Combinations of Machine and Patient Control over Phase Variables

	Phase Variable		
Breath Type	Trigger	Limit	Cycle
Mandatory	Machine	Machine	Machine
Assisted	Patient	Machine	Machine
Supported	Patient	Machine	Patient
Spontaneous	Patient	Patient	Patient

Machine-Cycled Breaths—*Mandatory breath*: a breath that is triggered, limited, and cycled by the ventilator (machine performs all ventilatory work).

Machine-Cycled Breaths—*Assisted breath*: a breath that is triggered by the patient and limited and cycled by the ventilator (patient performs only triggering work while the ventilator performs the remainder).

Patient-Cycled Breaths—*Supported breath*: a breath that is triggered by the patient, limited by the ventilator, and cycled by the patient (patient performs the triggering work and then interacts with the ventilator to perform a variable amount of the remaining work).

Patient-Cycled Breaths—*Spontaneous breath*: a breath that is triggered, limited,* and cycled by the patient (patient performs all of the ventilatory work).

The conditional variable is the variable (or combination of variables) that the ventilator's control logic examines before delivering a breath. The status of the conditional variable determines which of two or more breath patterns is selected.

The relationship between the various possible breath types and the conditional variables is the mode of ventilation. Table 2 applies the described classification system to commonly used modes of ventilatory support.

Support Features of a Mechanical Ventilator

A mechanical ventilator is a life support system designed to replace or support normal lung function. A variety of features exist that are designed to permit proper management of the patient in respiratory failure. Consensus recommendations for these features were determined and grouped according to the clinical setting (Table 3).

---

* When a demand-valve system is used, limiting of a spontaneous breath is, strictly speaking, controlled by the ventilator because flow is provided to maintain inspiratory pressure equal to expiratory pressure in an attempt to mimic 'normal' unassisted or unsupported breathing.

Table 2. Classification of Commonly Used Modes of Ventilation

Mode (Common Names)*	Breath Type												Conditional Variable
	Mandatory			Assisted			Supported			Spontaneous			
	Trigger	Limit	Cycle	Trigger	Limit	Cycle	Trigger	Limit	Cycle	Trigger	Limit	Cycle	
CMV/ VCV	Time	Flow	Volume†	–	–	–	–	–	–	–	–	–	–
ACV	Time	Flow	Volume†	Patient‡	Flow	Volume†	–	–	–	–	–	–	Patient effort/time
IMV	Time	Flow	Volume†	–	–	–	–	–	–	Patient‡	Pressure¶	Pressure¶	–
SIMV	Time	Flow	Volume†	Patient‡	Flow	Volume†	–	–	–	Patient‡	Pressure	Pressure	Patient effort/time
PCV	Time	Pressure	Time	–	–	–	–	–	–	–	–	–	–
PACV	Time	Pressure	Time	Patient‡	Pressure	Time	–	–	–	–	–	–	Patient effort/time
PIMV	Time	Pressure	Time	–	–	–	–	–	–	Patient‡	Pressure	Pressure	–
PSIMV	Time	Pressure	Time	Patient‡	Pressure	Time	–	–	–	Patient‡	Pressure	Pressure	Patient effort/time
APRV	Time	Pressure	Time	–	–	–	–	–	–	Patient‡§	Pressure	Pressure	–
Assist APRV	Time	Pressure	Time	Patient‡	Pressure	Time	–	–	–	Patient‡§	Pressure	Pressure	Patient effort/time
PSV	–	–	–	–	–	–	Patient	Pressure	Flow#	–	–	–	–
MMV	Time	Flow	Volume†	Patient‡	Flow	Volume†	–	–	–	Patient‡	Pressure	Pressure	Minute volume

* CMV/VCV = controlled mechanical ventilation/volume-controlled ventilation; ACV = assist-control ventilation; IMV = intermittent mandatory ventilation; SIMV = synchronized IMV; PCV = pressure-controlled ventilation; PACV = pressure ACV; PIMV = pressure IMV; PSIMV = pressure SIMV; APRV = airway pressure-release ventilation; PSV = pressure support ventilation; MMV = mandatory minute ventilation.

† Cycling can also be due to set inspiratory time in the setting of a fixed flow, with or without a pause.

‡ May be patient-generated pressure or flow in the circuit.

§ Allows spontaneous breathing during both the mandatory inspiratory and expiratory time.

Flow reflects interaction of patient effort respiratory system impedance, and ventilator flowrate.

¶ Pressure-limited only on demand-valve systems in which the ventilator limits and cycles to maintain constant airway pressure (thus, this applies to all modes in this column).

Table 3. Essential,[*] Recommended, and Optional Features of a Positive Pressure Ventilator for Mechanical Ventilatory Support via the Controlled Airway

	Principal Ventilator Application		
	Critical Care	Transport	Home Care
Clinician-Set Features			
Positive pressure tidal breaths	Essential	Essential	Essential
Mandatory rate	Essential	Essential	Essential
Flow or I:E or inspiratory time	Essential	Recommended	Recommended[‡]
Expiratory pressure (PEEP)	Essential	Essential	Optional
F_{DO_2}[†] to 1.0	Essential	Essential	Optional
Patient-Interactive Features			
Patient spontaneous breath (eg CPAP, IMV)	Essential	Recommended	Optional
Breath-triggering mechanism (flow or pressure sensors to initiate a ventilator breath)	Essential	Recommended	Recommended[‡]
Flow-timing interaction (eg, pressure support)	Recommended	Optional	Optional
Feedback Control (eg, mandatory minute ventilation)	Optional	Optional	Optional

[*] Essential = considered to be necessary for safe and effective operation in the majority of patients in the specified setting; recommended = considered to be necessary for optimal management of virtually all patients in the specified setting; Optional = considered to be possibly useful in limited situations but not necessary for a majority of patients in the specified setting.

[†] F_{DO_2} = oxygen concentration delivered by device; $F_{DO_2} = F_{IO_2}$ when patient demand (inspiratory flowrate) is met.

[‡] Essential feature if patient has intact ventilatory drive and respiratory muscles or possibility of partial or complete ventilator independence is anticipated.

Triggering

Gas flow from a mechanical ventilator can be triggered by patient effort. This effort is generally sensed either as a pressure drop (pressure-triggering) or as a gas flow into the patient (flow-triggering). Imposed work from triggering is due both to lack of sensitivity and responsiveness of the demand valves and to the post-triggering flow-delivery algorithm. The latter appears to be the more important. Flow-triggering measurably reduces both of these imposed loads as compared to pressure-triggering. However, the addition of a small level of pressure support (5 cm H_2O) has been reported to compensate for the work imposed during pressure-triggering and to make imposed loads comparable during both triggering strategies. Trigger-sensing sites (eg, patient Y, inspiratory limb, or expiratory limb) are comparable throughout the ventilator circuitry.

Heating and Humidification of Inspired Gases

When the upper airway is bypassed by tracheal intubation, inspired gases must be artificially conditioned to preserve airway integrity. Consensus was reached that both passive and active conditioning are acceptable, depending upon the patient's condition, duration of intubation, and the performance characteristics of the specific conditioning device.

A reasonable goal for humidification systems is an absolute humidity of 25-35 mg H_2O/L of ventilation. Heated humidifiers meeting the ASTM recommendations for a minimal output of 30 mg H_2O/L provide this. In addition, many heat-and-

Table 4. Measurements of Lung Mechanics and Airway Pressures Useful for Monitoring

Variable	Description	Measuring Technique
Effective compliance (C_{eff})	The reciprocal of the elastic property of the patient-ventilator system (mL/cm H_2O)	Mandatory (ie, passive inspiration and expiration) breath (V_T); end-inspiratory pause of at least 1 s with pressure stable within 0.5 cm H_2O over 2 readings at least 10 ms apart ($P_{plateau}$); corrected for tubing compression. Calculation: $C_{eff} = V_T/(P_{plateau} - \text{total PEEP})$.
Inspiratory resistance	Inspiratory resistive component of patient-ventilator system impedance (cm $H_2O \cdot s \cdot L^{-1}$)	Mandatory (ie, passive inspiration and expiration) breath (V_T) with fixed flow over fixed time (t_I); end-inspiratory pause as described for C_{eff}. Calculation: $R_I = \dfrac{P_{peak} - P_{plateau}}{V_T/t_I}$.
Expiratory resistance	Expiratory resistive component of patient ventilator system (cm $H_2O \cdot s \cdot L^{-1}$)	Mandatory (ie, passive inspiration and expiration) breath (V_T); end-inspiratory pause as described for C_{eff}. Calculation: $R_E = \dfrac{P_{plateau} - PEEP_{total}}{\text{flow at onset of exhalation}}$.
Mean airway pressure	Average airway pressure over a respiratory cycle	Mean airway pressure should be reported over a time period that includes a representative number of machine- and patient-cycled breaths.
Maximal inspiratory pressure (MIP)	Maximal inspiratory pressure generated by patient, against closed circuit	One-way valve allowing expiration; expiratory hold of 15-20 s
Intrinsic PEEP (auto-PEEP)	Positive end-expiratory alveolar pressure due to inadequate expiratory time, dynamic airway collapse, or both	Auto-PEEP measurement is clinically important but may be difficult during spontaneous or assisted breathing. It is recommended that the ventilator be equipped with an expiratory hold control to facilitate manual determination of auto-PEEP by airway occlusion as close to the proximal airway as possible; circuit pressures should stabilize within 0.5 cm H_2O between 2 consecutive readings at least 10 ms apart. Measurement reflects total PEEP but tends to underestimate the intrinsic component because of pressure equilibration in compliant circuitry

moisture exchangers (HME) or hygroscopic condenser humidifiers (HCH) meet this goal and can be used to supply heat and humidity to mechanically ventilated patients. Patient response to heat and humidification therapy should be assessed regularly, with particular attention to sputum consistency. If patients develop increased work of breathing or marked increase in secretion viscosity, consideration should be given to the utilization of a system providing higher humidity.

Monitoring and Alarms

Monitoring

A wide variety of electronic, pneumatic, and ventilator circuit functions can be monitored by the ventilator. However, the variables necessary for clinical decision making are pressure, flow, and volume, monitored in the ventilator circuitry.

Pressure monitoring should conform to ASTM[1] and International Organization for Standardization (ISO) standards[2] for accuracy, pressure ranges, and labeling.

Volume or flow monitoring should be done either at the patient Y or in the inspiratory or expiratory limb of the circuit. The accuracy and resistance of these devices should comply with ASTM[1] and ISO[2] standards.

The classic measurements of *respiratory mechanics* are made using sophisticated equipment under controlled conditions. These same measurements can be approximated in the intensive care unit using existing ventilator technology but must be accomplished under specific circumstances. The measurements of respiratory function that may be useful in the mechanically ventilated patient, when accomplished by the ventilator in the manner described, are outlined in Table 4. Essential and recommended variables to be monitored are grouped according to clinical setting (Table 5).

Alarms

Alarms should warn of events. These events may be malfunctions of the ventilatory support system (especially the circuit) or they may be physiologic or pathologic changes in the patient that affect the patient-ventilator interface. These events can be of varying importance and can be classified as such (Table 6): Level 1—immediately life-threatening if left unattended for even short periods of time; Level 2—potentially life-threatening if left unattended for longer periods of time; and Level 3—nonventilator events that are not likely to be life-threatening but a possible source of patient harm if not addressed. The alarm system should conform to ASTM[1] and ISO[2] standards and should provide a warning signal(s) if the function of the ventilator deviates from the control settings by more than preset levels. Events can be detected by various monitors at a number of sites (Table 7). It should not be possible to silence alarms for more than 2 minutes. Depending upon the clinical situation, the need for alarms varies, as shown in Table 8.

Digital Electronic Communication

The purpose of a mechanical ventilator is to support ventilation by supplying gas and pressure. There is no essential role for digital communications in the primary function of the mechanical ventilator; however, automatic capture of ventilator data should make respiratory care charting both more accurate and more timely, in the future. In order to provide effective digital electronic communication, an optimal algorithm for automated respiratory care charting should be developed.

Ventilator Management and Orders for Mechanical Ventilation

Authority to order mechanical ventilation varies in different practice settings. However, actual management of ventilatory support in all settings requires a certain level of expertise and typically involves participation by more than one individual. Ventilatory support should be managed by persons with appropriate training and demonstrated skills in both its medical and its technical aspects. The competence of such individuals should include but not necessarily be limited to

1. initiation of ventilatory support,
2. weaning and extubation,
3. patient monitoring related to ventilatory support,
4. selection and adjustment of ventilatory mode, inspired oxygen concentration, inspiratory pressure, end-expiratory pressure, tidal volume, and breath rate.

Ventilator orders by physicians may take the form of actual machine settings, specified outcomes (eg, target ranges for pH, P_{O_2}, or total breath rate), or the initiation of management protocols.

Table 5. Essential,* Recommended, and Optional Variables To Be Monitored on Mechanical Ventilators†

Variable	Principal Ventilator Application		
	Critical Care	Transport	Home Care
Pressure			
P_{peak}	Essential	Essential	Essential
P_{mean}	Essential	Optional	Optional
$P_{plateau}$	Essential	Optional	Optional
PEEP (set)	Essential	Essential	Optional‡
Intrinsic PEEP (auto-PEEP)	Recommended	Optional	Optional
Volume			
V_T expired machine	Essential	Recommended	Optional
$\dot{V}_E$ machine	Essential	Optional	Optional
V_T expired spontaneous	Essential	Recommended	Optional
$\dot{V}_E$ spontaneous	Essential	Optional	Optional
V_T inspired spontaneous	Recommended	Optional	Optional
Timing			
Flow mechanical	Recommended	Optional	Optional
Flow spontaneous	Optional	Optional	Optional
I-E ratio	Essential	Recommended	Optional
Rate mechanical	Essential	Recommended	Optional
Rate spontaneous	Essential	Recommended	Optional
Gas Concentration			
F_{DO_2}§	Essential	Optional‡	Optional‡
Lung Mechanics			
Effective compliance	Optional	Optional	Optional
Inspiratory airways resistance	Optional	Optional	Optional
Expiratory airways resistance	Optional	Optional	Optional
Maximal inspiratory pressure	Optional	Optional	Optional
Circuit Characteristics			
Tubing compliance	Recommended	Optional	Optional

---

*Essential = considered to be necessary for safe and effective operation in the majority of patients in the specified setting; Recommended = considered to be necessary for optimal management of virtually all patients in the specified setting; Optional = considered to be possibly useful in limited situations but not necessary for a majority of patients in the specified setting.

†Monitors need not be intergral part of ventilator.

‡Essential if feature is used on a specific patient.

§F_{DO_2} = oxygen concentration delivered by device; F_{DO_2} = F_{IO_2} when patient demand (inspiratory flowrate) is met.

Orders for ventilatory support may authorize appropriate individuals (eg, qualified respiratory care practitioners) to independently adjust the degree and form of such support, in accordance with guidelines established for the clinical setting in which the order is given.

Protocols for ventilator management should be tailored to specified clinical situations such as post-

Table 6. Priorities for Mechanical Ventilator Alarms

Priority	Life-Threatening	Immediate Response Required	Redundant	Alarm Type
Level 1	Yes, immediately	Yes	Yes	Loud audible & visual
Level 2	Yes, potentially	Yes	No	Soft audible & visual
Level 3	No	No	No	Visual

operative ventilation, application of PEEP, or weaning. They should be formulated by qualified personnel and be appropriate for the specific practice setting. Initiation of established protocols is the preferred form for ventilator orders in the majority of patients requiring mechanical ventilation.

A record of ventilatory orders, including the specifics of protocols authorized by such orders, should be continuously available at the bedside. All ventilator orders should be part of the permanent hospital record and should be documented appropriately.

Table 7. Events and Monitoring Sites for Ventilator Alarms

Event	Possible Monitoring Site
Level 1	
Power failure (including when battery in use)	Electrical control system*
Absence of gas delivery (apnea)	Circuit pressures,* circuit flows, timing monitor, CO_2 analysis
Loss of gas source	Pneumatic control system*
Excessive gas delivery	Circuit pressures,* circuit flows, timing monitor
Exhalation valve failure	Circuit pressures, circuit flows, timing monitor
Timing failure	Circuit pressures, circuit flows, timing monitor
Level 2	
Battery power loss (not in use)	Electrical control system*
Circuit leak*	Circuit pressures,* circuit flows
Blender failure	F_{IO_2} sensor
Circuit partially occluded	Circuit pressures, circuit flows
Heater/humidifier failure	Temperature probe in circuit
Loss of/or excessive PEEP	Circuit pressures
Autocycling	Circuit pressures, circuit flows
Other electrical or preventive subsystem out of limits without immediate overt gas delivery effects	Electrical and pneumatic systems monitor
Level 3	
Change in central nervous system drive	Circuit pressures, circuit flows, timing monitor
Change in impedances	Circuit pressures, circuit flows, timing monitor
Intrinsic PEEP (auto) > 5 cm H_2O	Circuit pressures, circuit flows

---

*Alarms currently defined in the ISO[1] and ASTM[2] standards.

Table 8. Essential,* Recommended, and Optional Alarms for Mechanical Ventilators†

	Principal Ventilator Application		
Level‡	Critical Care	Transport	Home or Skilled Nursing Facility
Level 1	Essential	Essential	Essential§
Level 2	Essential	Essential	Recommended
Level 3	Recommended	Optional	Optional

---

*Essential = considered to be necessary for safe and effective operation in the majority of patients in the specified setting; recommended = considered to be necessary for optimal management of virtually all patients in the specified setting; optional = considered to be possibly useful in limited situations but not necessary for a majority of patients in the specified setting.

† Alarms need not be integral components of the ventilator.

‡ Levels are defined in Table 6.

§ Redundancy is required only if ventilator is providing total support.

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Faculty:

Richard D Branson RRT, Cincinnati OH
Robert L Chatburn RRT, Cleveland OH
Thomas D East PhD, Salt Lake City UT
Robert M Kacmarek PhD RRT, Boston MA
Neil R MacIntyre MD, Durham NC
John J Marini MD, St Paul MN
David J Pierson MD, Seattle WA
Catherine Sassoon MD, Long Beach CA
Maire P Shelly MB ChB FFARCS, Manchester UK
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David Burns MD, San Diego CA
T James Gallagher MD, Gainesville FL
John Overman BSEE, Kansas City KS
Mark H Sanders MD, Pittsburgh PA
Arthur S Slutsky MD, Toronto, Ontario, Canada

Planning-Writing Committee:

Richard D Branson RRT, Cincinnati OH
Pat Brougher RRT, Dallas TX
Robert L Chatburn RRT, Cleveland OH
Thomas D East PhD, Salt Lake City UT
John J Marini MD, St Paul MN
Neil R MacIntyre MD, Durham NC

Classification of Mechanical Ventilators

Robert L Chatburn RRT

I. Introduction
II. Basic Concepts
III. The Theoretical Basis for Ventilator Classification
IV. Application of Principles of Classification System
  A. Control Variables and Waveforms
  B. Phase Variables
  C. Conditional Variables
  D. Output Waveforms
  E. Ventilator-Alarm Systems
V. How To Speak Machine Language

> Error of opinion may be tolerated where reason is left free to combat it.
>
> *Thomas Jefferson (1743-1826)*

Introduction

A good ventilator classification scheme describes how a given ventilator works in general terms, but with enough detail to allow one to distinguish one particular ventilator from others. Classification facilitates description by focusing on key attributes or characteristics in a logical and consistent manner. A clear description allows us to quickly assess new facts in relation to previous knowledge. Learning the operation of a new device or describing it to others then becomes much easier. Understanding how the ventilator operates, we can then anticipate appropriate ventilator management strategies for particular clinical situations.

Ventilator classification has been attempted many times. I have noted elsewhere[1] the problems encountered in the past. Typical examples of these problems can be seen in the recent ASTM document entitled *Standard Specification for Ventilators Intended for Use in Critical Care.*[2] An appendix in that standard is devoted to ventilator classification. Four major categories of problems with past classification systems can be identified.

- They are outdated. Most are variations on the classic work of Mushin et al first published in 1959.[3] The most recent version of this text is more than 12 years old. Mushin's classification system is based on archetypical mechanical mechanisms that are largely irrelevant today. It also emphasizes differences in the "generated" or driving pressure that ventilators can develop. This too is outdated because most third-generation ventilators are driven by the standard 50-psi gas source piped into intensive care units.

Mr Chatburn is Director, Pediatric Respiratory Care, Rainbow Babies and Childrens Hospital, and Instructor, Department of Pediatrics, Case Reserve University–Cleveland, Ohio.

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Reprints: Robert L Chatburn RRT, Respiratory Care Department, Rainbow Babies & Childrens Hospital, 2074 Abington Road, Cleveland OH 44106.

- They are contradictory. For example, the word cycle has been used to mean two different things (ie, to designate both the beginning and the end of inspiration).

- They are vague. For example, the words mandatory and spontaneous are used but never defined.

- They lack appropriate detail. For example, most systems do not distinguish between situations in which inspiration ends because a preset volume has been delivered and the situation in which inspiration ends only when a preset time has elapsed even though the preset volume has been delivered (ie, an inspiratory pause maneuver).

To avoid these problems, a classification scheme should have the following characteristics:

- Be based on a theoretical framework. The system should identify a small set of basic, related physical principles (eg, a mathematical model) that can be applied to all ventilator types and that adequately describes the interaction between the ventilator and the patient. This avoids a collection of loosely related facts that must be memorized.

- Be consistent. It should avoid double meanings by introducing new terminology as necessary. The new terminology should be consistent with terminology in other disciplines (eg, mathematics and engineering). Old terminology should be retained where possible to avoid undue confusion.

- Be specific. All terms used to categorize ventilator operation should be explicitly defined. Criteria that are necessary and sufficient to distinguish between ventilator characteristics should be clearly stated.

- Provide appropriate detail, identifying clinically relevant situations while avoiding unnecessary complexity.

I believe the proposed system incorporates those characteristics.

Basic Concepts

A ventilator is simply a machine—a system of related elements designed to alter, transmit, and direct applied energy in a predetermined manner to perform useful work.[4] We put energy into the ventilator in the form of electricity (energy = volts × amperes × time) or compressed gas (energy = pressure × volume). That energy is transmitted or transformed (by the ventilator's drive mechanism) in a predetermined manner (by the control circuit) to augment or replace the patient's muscles in performing the work of breathing (the desired output). This concept of mechanical ventilators suggests a basic framework for classification: power input, power transmission or conversion, control scheme, and output (pressure, volume, and flow waveforms).

An outline format provides the flexibility to add as much detail about a given ventilator as desired.[1]

I. Power Input
  A. Pneumatic
  B. Electric
    1. Alternating current (AC)
    2. Direct current (DC; battery)

II. Power Transmission or Conversion (drive mechanism)
  A. External Compressor
  B. Internal Compressor
    1. Motor and linkage
      a. Compressed gas—direct
      b. Electric motor—rotating crank and piston rod
      c. Electric motor—rack and pinion
      d. Electric motor—direct
  C. Output Control Valves
    1. Pneumatic diaphragm
    2. Pneumatic poppit valve
    3. Electromagnetic poppit valve
    4. Electromagnetic proportional valve

III. Control Scheme
  A. Control Circuit
    1. Mechanical
    2. Pneumatic
    3. Fluidic
    4. Electric

    5. Electronic
  B. Control Variables and Waveforms
    1. Pressure
    2. Volume
    3. Flow
    4. Time
  C. Phase Variables
    1. Trigger Variable
    2. Limit Variable
    3. Cycle Variable
    4. Baseline Variable
  D. Conditional Variables

IV. Output
  A. Pressure Waveforms
    1. Pulse (ie, rectangular)
    2. Exponential rise
    3. Sinusoidal
    4. Oscillating
  B. Volume Waveforms
    1. Ramp
    2. Sinusoidal
  C. Flow Waveforms
    1. Pulse (ie, rectangular)
    2. Ramp
      a. Ascending
      b. Descending
    3. Sinusoidal

V. Alarm Systems
  A. Input Power Alarms
    1. Loss of electric power
    2. Loss of pneumatic power
  B. Control Circuit Alarms
    1. General systems failure (ventilator inoperative)
    2. Incompatible ventilator settings
    3. Inverse inspiratory-to-expiratory-time ratio
  C. Output Alarms
    1. Pressure
      a. High and low peak airway pressure
      b. High and low mean airway pressure
      c. High and low baseline pressure (PEEP or CPAP)
      d. Failure of airway pressure to return to baseline within a specified period
    2. Volume (low tidal volume)
    3. Flow (low minute ventilation)
    4. Time
      a. High or low ventilatory frequency
      b. Long or short inspiratory time
      c. Long or short expiratory time (long expiratory time = apnea)
    5. Inspired gas
      a. High or low inspired gas temperature
      b. High or low F_{IO_2} (F_{DO_2})

A discussion of input power sources and power conversion/transmission is beyond the scope of this article. However, control schemes and ventilator output (ie, pressure, volume, and flow) are explored in detail. The classification system is also applied to alarms.

The Theoretical Basis for Ventilator Classification

To understand how a machine can be controlled to replace or supplement the natural function of breathing, we need to understand something about the mechanics of breathing. The study of mechanics deals with forces, displacements, and the rate of change of displacement. In physiology, force is measured as pressure (pressure = force ÷ area), displacement is measured as volume (volume = area × displacement) and the relevant rate of change is measured as flow (eg, average flow = Δvolume ÷ Δtime; instantaneous flow = dv/dt, the derivative of volume with respect to time). Specifically, we are interested in the pressure necessary to cause a *flow* of gas to enter the airway and increase the *volume* of the lungs.

The study of respiratory mechanics is essentially the search for simple but useful models of respiratory-system mechanical behavior. Figure 1 depicts the respiratory system in a graphic model and in a mathematical model based on the graphic model. Measurable variables (ie, pressure, volume, and flow) are related by a mathematical model known as the equation of motion for the respiratory system:[5]

$$\text{muscle pressure} = \text{ventilator pressure} = \text{volume/compliance} + (\text{resistance})\,(\text{flow}). \qquad [1]$$

In this simplified form, muscle pressure is the 'imaginary' transrespiratory pressure (ie, airway

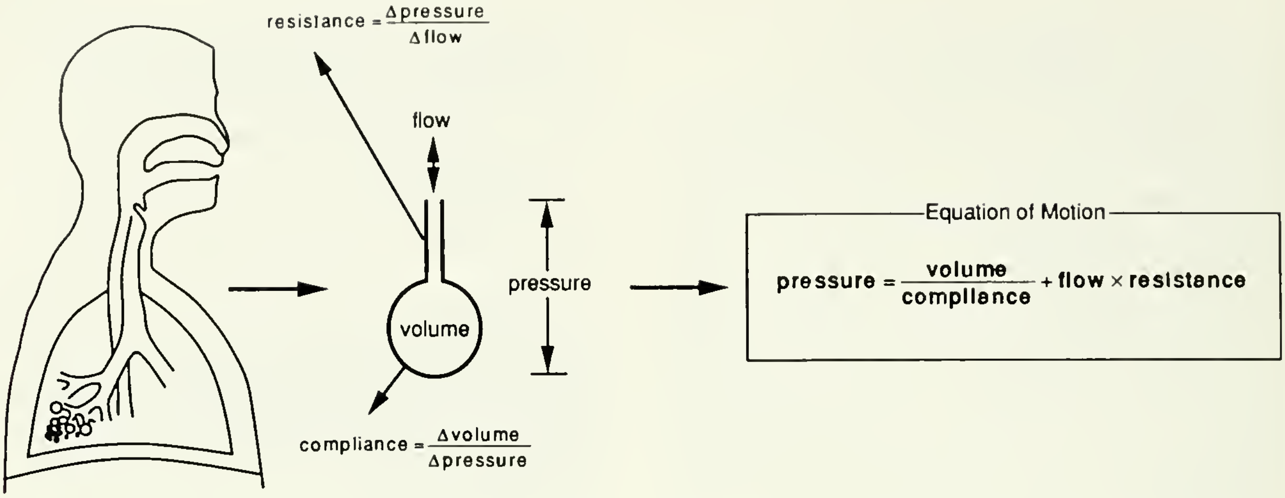
resistance = Δpressure / Δflow
flow
pressure
volume
compliance = Δvolume / Δpressure
Equation of Motion
pressure = volume / compliance + flow × resistance

Fig. 1. The study of respiratory system mechanics is based on graphical and mathematical models. Specifically, the respiratory system is often conceived as a single resistance connected to a single compliance. The mechanical behavior of this system is described by a mathematical model called the equation of motion for the respiratory system. In this model, pressure, volume, and flow are variables (ie, functions of time) while resistance and compliance are constants.

pressure minus body surface pressure) generated by the ventilatory muscles to expand the thoracic cage and lungs. Muscle pressure is said to be imaginary because it is not directly measurable. Ventilator pressure is the transrespiratory pressure generated by the ventilator during inspiration. The combined muscle and ventilator pressure cause volume and flow to be delivered to the patient. Pressure, volume, and flow change with time and hence are variables. Compliance and resistance are assumed to remain constant and are called parameters, and their combined effect (ie, the impedance) constitutes the load experienced by the ventilator and ventilatory muscles. The term parameter may also refer to a particular aspect of a variable such as the peak or mean value.

Notice that if the patient's ventilatory muscles are not functioning, muscle pressure = 0, and the ventilator must generate all of the pressure required to deliver the tidal volume and inspiratory flow. On the other hand, a ventilator is not needed for normal, spontaneous breathing (ie, ventilator pressure = 0). In between these two extremes, an infinite number of combinations of muscle pressure (ie, patient effort) and ventilatory support are theoretically possible. These combinations suggest a great many so-called modes of ventilation for partial ventilatory support.

It is important to remember that pressure, volume, and flow are measured relative to their baseline values. Thus, for example, ventilator pressure actually means airway pressure minus baseline pressure (ie, ventilator pressure = P_{aw} − CPAP). Clinicians are often unaware of this distinction and believe, for example, that a patient breathing spontaneously on CPAP is somehow aided by the ventilator. In fact, ventilator pressure (from the equation of motion) on CPAP is zero (ie, P_{aw} = CPAP, so ventilator pressure = CPAP − CPAP = 0), and the patient must generate all of the work of breathing, using the ventilatory muscles.

This type of analysis also suggests the proper use of another frequently misunderstood term—assist. One dictionary defines assist as "to help; to aid; to give support . . .." From the perspective of the equation of motion, whenever airway pressure (ie, ventilator pressure) rises above baseline during inspiration, the ventilator does work on the patient: work = Δpressure × Δvolume. Thus, the breath is assisted or helped. This was the original intent of the so-called Assist Mode on early mechanical ventilators: The patient had only to initiate the breath and the ventilator did the rest of the work. Unfortunately, many people have come to associate the word assist with a very narrow definition: a specific mode of ventilation in which the patient can trigger an inspiration with a preset tidal volume. This is because early ventilators had a knob that could be turned to an Assist or Assist/Control position to deliver this pattern. Now it is difficult for people to view newer modes of ventilation, such as Pressure Support or Proportional Assist as forms of assisted ventilation in which the ventilator contributes some of the work of breathing.

The equation of motion is of central importance in classifying ventilators. The behavior of the variables in the equation provide a model for the behavior of a ventilator connected to a patient. To understand what I mean by the behavior of variables in an equation, consider the simple equation:

$$\text{pressure} = (\text{resistance})\,(\text{flow}), \qquad [2]$$

where pressure and flow are variables and resistance is a constant.

Now, if we make flow a linear function of time, then we have specified that its form is:

$$\text{flow} = (\text{A})\,(\text{time}), \qquad [3]$$

where A is a constant.

How will pressure behave once the form of flow has been specified? We find out by substituting Equation 3 into Equation 2:

$$\text{pressure} = (\text{resistance})\,(\text{A})\,(\text{time}). \qquad [4]$$

But because resistance and A are both constants, they can be replaced by another constant, B:

$$\text{pressure} = (\text{B})\,(\text{time}). \qquad [5]$$

Equation 5 has the same form as Equation 3, which tells us that pressure, just like flow, is a linear function of time. In mathematical terms, flow is called the independent variable because we specified its form and this form is independent of anything else in the equation. Pressure is considered to be the dependent variable because its form depends on the form of the independent variable, flow, and the constant B. Of course, we could have first specified the form of pressure and then observed the resultant form for flow. In this case, pressure would have been considered the independent variable and flow the dependent variable.

We can extend this analysis to the equation of motion (Equation 1). It follows that in this equation, the form of any one of the three variables (ie, pressure, volume, or flow) can be predetermined, making it the independent variable and making the other two dependent variables. This is precisely analogous to the way ventilators operate. For example, when the Siemens Servo 900C is set to the Pressure Control mode, it controls the shape of the airway pressure waveform (it has an approximately rectangular shape). Thus, pressure is the independent variable, and the shape of the volume and flow waveforms both depend on the shape of the pressure waveform and also on the resistance and compliance of the respiratory system. (The volume and flow waveforms determine clinically relevant parameters such as tidal volume and peak inspiratory flowrate.) On the other hand, in the Volume Control mode, we can specify that flow has a rectangular waveform. This makes flow the independent variable, and the shapes of the volume and pressure waveforms depend on the shape of the flow waveform as well as on resistance and compliance. (The volume and pressure waveforms determine clinically relevant parameters such as tidal volume, peak inspiratory pressure, and mean airway pressure.)

We now have a theoretical basis for classifying ventilators as either pressure-, volume-, or flow-controllers. The necessary and sufficient criteria for determining which variable is controlled (ie, which variable is the independent variable) are illustrated in Figure 2. Note that if the waveforms for all three variables are not predetermined (ie, none of the variables can be considered independent), then the ventilator is considered to control only the timing

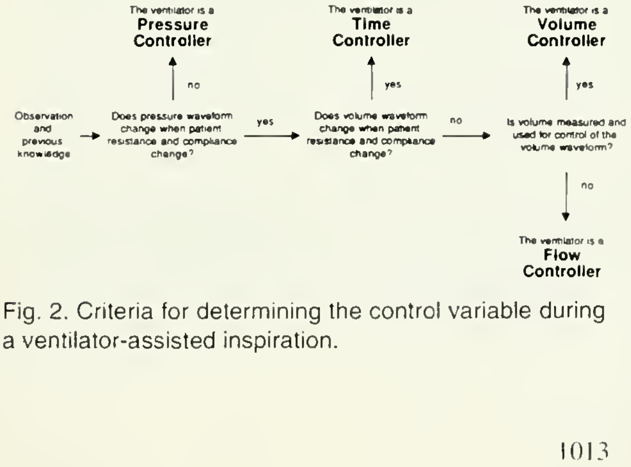
The ventilator is a
Pressure Controller
no
The ventilator is a
Time Controller
yes
The ventilator is a
Volume Controller
yes
Observation and previous knowledge
Does pressure waveform change when patient resistance and compliance change?
yes
Does volume waveform change when patient resistance and compliance change?
no
Is volume measured and used for control of the volume waveform?
no
The ventilator is a
Flow Controller

Fig. 2. Criteria for determining the control variable during a ventilator-assisted inspiration.

of the inspiratory and expiratory phase and is called a time-controller. In at least one case (ie, proportional assist ventilation[6]), the ventilator does not predetermine the time course of any variable, but for convenience we will consider it a time-controller by default.

The ventilator's control scheme may be sophisticated, with the entire shape of the waveform predetermined during inspiration. Many third-generation ventilators can do this and even offer a choice of waveforms. On the other hand, the control scheme may be quite simple, with only one waveform parameter, such as the peak or mean value, controlled during inspiration. The most common example is the way that second-generation infant ventilators control pressure simply by limiting the peak inspiratory value.

For each control variable, a limited number of waveforms are commonly used by current ventilators, although almost any waveform is possible. These waveforms can be idealized as shown in Figure 6 and have been grouped into four basic categories: *pulse, exponential, ramp,* and *sinusoidal.* These terms are consistent with the terms used in mathematics and electrical engineering to describe these waveforms. (Note that a pulse waveform is theoretically impossible for volume because volume cannot change instantaneously from zero to some preset value as pressure and flow can.)

Application of Principles of Classification Sytstem

Control Variables and Waveforms

Pressure-Controllers—If the control variable is pressure, the ventilator can control either the airway pressure (causing it to rise above body surface pressure for inspiration) or the pressure on the body surface (causing it to fall below airway opening pressure for inspiration). This is the basis for classifying ventilators as being either positive or negative pressure types. For example, the Newport Wave ventilator can be classified as a positive-pressure-controller that generates a rectangular pressure waveform, and the Emerson Iron Lung as a negative-pressure-controller that produces a quasi sinusoidal pressure waveform.

The equation of motion tells us that if the ventilator is an ideal *pressure controller* then the left side of the equation (ie, ventilator pressure as a function of time) is determined by the ventilator settings (eg, peak inspiratory and end-expiratory pressure) and is unaffected by changes in parameter values on the right side (ie, compliance and resistance).

Volume-Controllers—If the pressure waveform varies as the load changes, we then examine the volume waveform. However, the observation that the volume waveform remains unchanged is a necessary but not a *sufficient* condition to warrant the classification of volume-controller because the same holds true for a flow-controller. The reason is that once the volume waveform is specified, the flow waveform is determined because they are functions of each other (ie, volume is the integral of flow, and flow is the derivative of volume). Therefore, if changes in compliance and resistance do not change the volume waveform, they do not affect the flow waveform, and vice versa.

To qualify as a *volume-controller* a ventilator must (1) maintain an approximately constant volume waveform in the face of a varying load and (2) measure volume and use the signal to control the volume waveform. Volume can be measured directly only by the displacement of a piston or bellows or similar device. With a piston or bellows, controlling the excursion of the device automatically controls the volume waveform. Two examples of this design approach are the Emerson 3MV and the Bennett MA-1. Alternatively, a volume signal can be derived by integrating a flow signal. Note that although some ventilators like the Siemens Servo 900C, the Bennett 7200, the Bear 5, and the Hamilton Veolar display volume readings, they all actually measure and control flow and calculate volume for displays. Thus, they are all flow-controllers unless they are operated in a pressure-control mode (eg, during pressure support ventilation). An examination of a ventilator's schematic diagrams and operator's manual should provide the information necessary to decide whether volume or flow is being measured.

Flow-Controllers—If the volume waveform remains unchanged when compliance and resistance are varied, and if volume is not directly measured or used as a feedback signal, the ventilator is classified as a flow-controller. For example, an infant ventilator becomes a flow-controller rather than a pressure-controller if the airway pressure does not reach the set pressure limit. However, the flowmeter is usually not pressure compensated, and its output varies slightly in the face of a changing load. In contrast, the Siemens Servo 900C is so-called because it uses servo control (measures flow and adjusts the output control valve [ie, the inspiratory scissors valve] accordingly). It can maintain a more consistent inspiratory flow waveform as the load changes.

Time-Controllers—If both pressure and volume waveforms are affected substantially by changes in lung mechanics, then the only form of control is that of defining the ventilatory cycle or alternating between inspiration and expiration. Therefore, the only variables being controlled are the inspiratory and expiratory times. This situation arises in some forms of high frequency ventilation in which even the designation of an inspiratory and expiratory phase becomes somewhat obscure.

Phase Variables

Once the control variable and the associated waveforms are identified, more detail can be obtained by examining the events that take place during a ventilatory cycle (ie, the period of time between the beginning of one breath and the beginning of the next). Mushin et al proposed that this time span be divided into four phases: the change from expiration to inspiration, inspiration, the change from inspiration to expiration, and expiration. In each phase a particular variable is measured and used to *start*, *sustain*, and *end* the phase. In this context, pressure, volume, flow, and time are referred to as *phase variables* (Fig. 2).

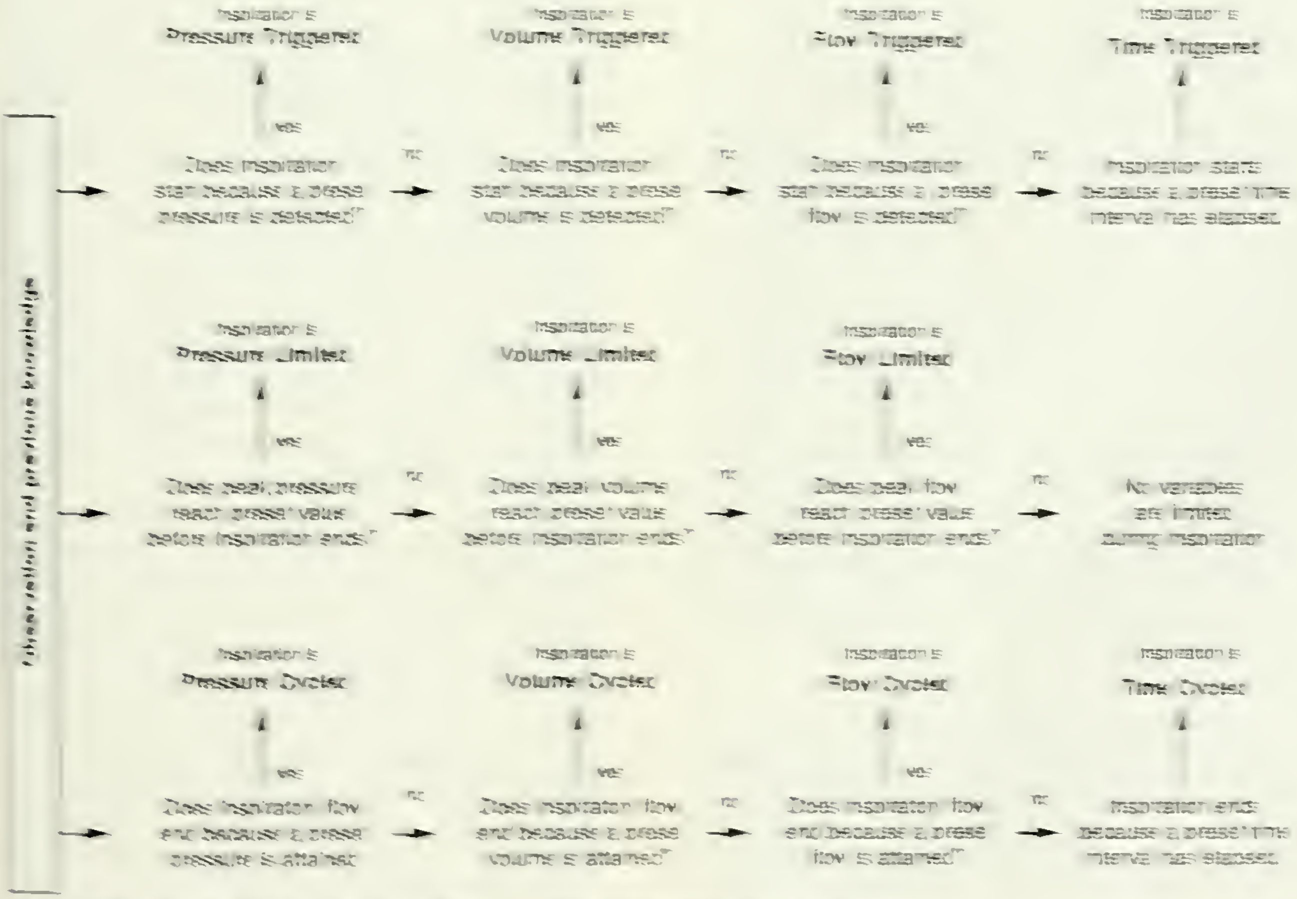
Inspiration is Pressure Triggered
yes
Does inspiration start because a preset pressure is detected?
no
Inspiration is Volume Triggered
yes
Does inspiration start because a preset volume is detected?
no
Inspiration is Flow Triggered
yes
Does inspiration start because a preset flow is detected?
no
Inspiration is Time Triggered
Inspiration starts because a preset time interval has elapsed
Inspiration is Pressure Limited
yes
Does peak pressure reach preset value before inspiration ends?
no
Inspiration is Volume Limited
yes
Does peak volume reach preset value before inspiration ends?
no
Inspiration is Flow Limited
yes
Does peak flow reach preset value before inspiration ends?
no
No variables are limited during inspiration
Inspiration is Pressure Cycled
yes
Does inspiration flow end because a preset pressure is attained?
no
Inspiration is Volume Cycled
yes
Does inspiration flow end because a preset volume is attained?
no
Inspiration is Flow Cycled
yes
Does inspiration flow end because a preset flow is attained?
no
Inspiration is Time Cycled
Inspiration ends because a preset time interval has elapsed

Fig. 2. Criteria for determining the phase variables during a ventilator-assisted breath.

Trigger Variables—All ventilators measure one or more of the variables associated with the equation of motion (ie, pressure, volume, flow, or time). Inspiration begins when one of these variables reaches a preset value. Thus, the variable of interest is considered an initiating or *trigger variable*. The most common trigger variables are time (ie, the ventilator initiates a breath according to a set frequency, independent of the patient's spontaneous efforts) and pressure (ie, the ventilator senses the patient's inspiratory effort in the form of a drop in baseline pressure and starts inspiration independent of the set frequency). Of course it may be possible to *manually trigger* inspiration. Indeed, it is possible to trigger inspiration using almost any type of signal. For example, the Infrasonics StarSync device triggers the ventilator in response to the infant's chest-wall movements during an inspiratory effort.

It is feasible for a ventilator to measure volume and flow changes caused by the patient's inspiratory efforts and use them to trigger inspiration, but the technology is more complex and is not commonly used. Triggering on flow has been shown to be more sensitive than triggering on pressure (at least with conventional patient circuits); hence, the patient has to do less work on the ventilator to obtain a breath.[9] On the other hand, the flow signal is prone to noise caused by movement of the patient circuit and turbulence from condensation in the circuit. This may lead to false triggering. Volume-triggering is less sensitive to such noise. Currently, only the Bennett 7200a can be flow-triggered and only the Dräger Babylog is volume-triggered.

The patient effort required to trigger inspiration is determined by the ventilator's *sensitivity*. Sensitivity is adjusted by changing the preset value of the trigger variable. For example, to make a pressure-triggered ventilator more sensitive, the trigger pressure can be adjusted from 5 cm H_2O to 1 cm H_2O below the baseline pressure.

Mandatory vs Spontaneous Breaths—Some ventilatory modes permit the patient to breathe spontaneously between mandatory breaths. But, here is an interesting dilemma: What is the difference between a mandatory and a spontaneous breath? Again, this is a problem born of technologic evolution. Taber's definition of spontaneous is "occurring . . . without apparent cause; voluntary."[10] It is clear, for example, that a patient breathes spontaneously when not connected to a ventilator—the patient controls the frequency and size of breaths and hypoventilates if apnea occurs.

Originally, ventilators controlled every breath. The idea that there could be such a thing as a spontaneous breath during mechanical ventilation arose when continuous-flow, intermittent mandatory ventilation (IMV) was introduced. Yet it is obvious, in the context of IMV, that mandatory breaths are triggered, or caused, by the ventilator, and spontaneous breaths are under voluntary control by the patient. Synchronized intermittent mandatory ventilation (SIMV) was created as a refinement of IMV in which the ventilator responds to patient breathing efforts in one of two ways depending upon when the effort occurs. A consideration of this mode complicates matters. If a patient inspiratory effort occurs at the right time, inspiration is pressure- (or flow-) triggered instead of time-triggered by the ventilator. The right time is usually referred to as a window or percentage of the ventilatory period (defined by the set SIMV frequency) during which the ventilator switches from patient-triggering to time-triggering. Thus, it seems that a breath may be either mandatory or spontaneous depending upon the coincidental occurrence of a patient effort within the timing window! A consideration of the pressure support mode raises the question of whether setting an inspiratory pressure limit constitutes a mandatory breath. We need more exact definitions of the terms mandatory and spontaneous. This will help us to understand what the ventilator is doing and will provide a basis for distinguishing between ventilatory modes.

To resolve these problems, we must recognize the importance of distinguishing between the types of breaths that can occur in any mode of ventilation. We must also use the simplest classification scheme to avoid unnecessary complexity. This means that we should try to define as few breath types as possible. One is clearly too few. Three or more are conceivable (eg, flow-controlled mandatory normal, flow-controlled mandatory sigh, and pressure-controlled spontaneous, or, perhaps, controlled, assisted, and spontaneous). Two seem about right—mandatory and spontaneous, based on the degree of control the patient has over the breath parameters. One dictionary defines *spontaneous* as ". . . acting . . . from a native internal . . . readiness, or tendency, without compulsion . . ." and *mandatory* as ". . . required; obligatory . . . containing a mandate" (ie, to control).[11] The following analysis should further illuminate the issue.

When the patient is not connected to the ventilator, the brain determines the trigger, limit, and cycle values in response to physiologic signals supplied by chemoreceptors and the Hering-Breuer reflex (ie, the brain initiates a breath, determines the peak inspiratory flowrate and waveform, and determines the size of the tidal volume). Ventilatory failure can be thought of as a derangement of these values due to problems with the physiologic sensors, the brain's ability to process the signals, failure of the ventilatory muscles, or pathology of the lungs. An ideal ventilator would sense the body's physiologic gas exchange needs just as the brain does and would provide assistance in proportion to the deficit in the trigger, limit, and cycle values.

Ventilatory failure can be partial or complete, giving rise to the need for partial or complete ventilatory support. In partial ventilatory failure, the body usually maintains a regular effort to trigger inspiration but has lost the capability to maintain adequate limit and cycle values (ie, inspiratory flowrate and tidal volume are too small). When the body tries to breathe spontaneously, the ideal ventilator would sense and preserve this ventilatory drive by allowing patient-triggering, patient-limiting, and patient-cycling of the assisted breath in exact proportion to the patient's need. Younes has recently described an experimental device that can do this.[6] (Unfortunately, current commercial devices have not evolved enough to allow this level of sophistication, and we are forced to add the judgment of the ventilator operator into the control loop.) In full ventilatory failure, the body no longer attempts to trigger, limit, or cycle inspiration, and the ventilator assume these functions. Thus, we can derive a clinically relevant definition of spontaneous and mandatory breaths.

Spontaneous breaths are those that the patient may both initiate and terminate with sufficient activity of the ventilatory muscles or by the passive characteristics of respiratory system compliance and resistance. If the ventilator determines either the start or end of inspiration, then the breath is considered to be *mandatory*. Figure 4 illustrates these definitions with an algorithm. Note that if the ventilator either time or volume cycles an inspiration, the breath is considered mandatory because it is terminated by the ventilator. If, however, the ventilator flow cycles an inspiration (as in the pressure support mode), it is not considered mandatory. The rate of decay of inspiratory flow is determined by the patient's lung mechanics and/or ventilatory muscle activity. Flow-cycling does not necessarily dictate either the inspiratory time or the tidal volume (particularly if there is ventilatory muscle activity). In other words, the ventilator attempts to match the patient's inspiratory demand, and it is really the patient who terminates the breath.

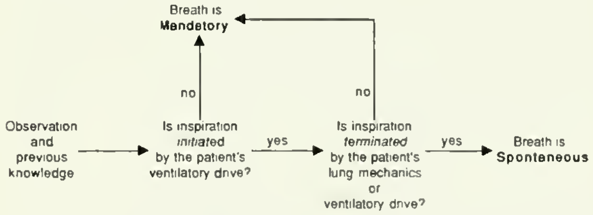
Breath is
Mandatory
no
no
Observation and previous knowledge
Is inspiration *initiated* by the patient's ventilatory drive?
yes
Is inspiration *terminated* by the patient's lung mechanics or ventilatory drive?
yes
Breath is
Spontaneous

Fig. 4. Algorithm defining spontaneous and mandatory breaths. In terms of current technology, the breath is mandatory if the breath is triggered according to a preset frequency or minimum minute ventilation or cycled according to a preset frequency or tidal volume. All other breaths are spontaneous.

amples: If the breath is triggered by the ventilator, then it is considered mandatory regardless of how it is limited and cycled. If the ventilator is set so that the patient may trigger a breath but the inspiratory flowrate and tidal volume are preset (eg, the Assist/Control or CMV mode), then the breath is mandatory because the patient cannot terminate the breath before or after the predetermined inspiratory time has elapsed. On the other hand, if the ventilator is set so that the patient may trigger a breath but the inspiratory pressure is limited and the breath is flow-cycled (eg, the pressure support mode), then the breath is *spontaneous* because the patient can still determine the instantaneous inspiratory flowrate and tidal volume, depending on the activity of the ventilatory muscles. To avoid confusion, it helps to perceive that all mandatory breaths are assisted in the sense (defined earlier) that the ventilator does work on the patient. Spontaneous breaths may be either assisted (eg, pressure support or proportional assist) or unassisted (eg, breathing on CPAP or not connected to a ventilator). Defining breaths in this way provides the basis for classifying modes of ventilation and may provide a simpler basis for selecting modes on the ventilator.

Limit Variable—During inspiration, pressure, volume, and flow increase above their end-expiratory values. If one (or more) of these variables rises no higher than some *preset* value, we refer to the variable as a *limit variable.* But we must distinguish the limit variable from the variable that is used to end inspiration (called a *cycle variable*). Therefore, we impose the additional criterion that *inspiration is not terminated because a variable has met its preset limit value.* In other words, a variable is limited if it increases to a preset value before inspiration ends. These criteria are illustrated in Figure 5.

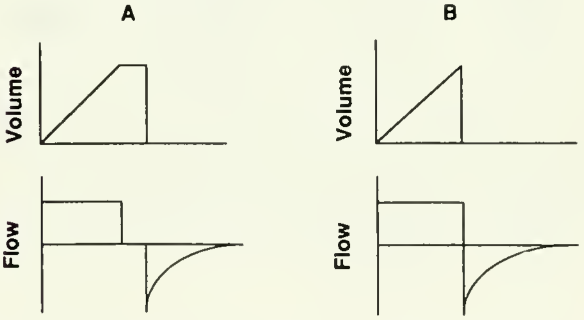

Fig. 5. This figure illustrates the importance of distinguishing between the terms limit and cycle. In A, both volume and flow are limited (because they reach preset values before end-inspiration) and inspiration is time-cycled (after the preset inspiratory pause time). In B, flow is limited but volume is not and inspiration is volume-cycled.

For example, peak pressure during conventional or high frequency oscillatory ventilation of neonates is frequently limited, but the preset pressure limit has nothing to do with the termination of inspiration. Rather, inspiration ends after a preset *time* interval, (ie, the preset inspiratory time). Another example: When an inspiratory pause is used on a ventilator like the Bear 5, the volume delivered to the patient is limited to a preset value, but inspiration does not end. Rather, inspiration ends when a preset time interval (in this case the pause time) has elapsed. One last example is the way peak inspiratory flowrate is often limited to a preset value (during ventilation of adults), but inspiration ends when a preset volume has been delivered.

Clinicians commonly use the terms limit and cycle interchangeably—a misuse of terms encouraged by some ventilator manufacturers who use the term limit to describe what happens when a pressure alarm threshold is met (ie, inspiration is terminated and an alarm is activated). The term cycle, as defined below, is more appropriate in this situation.

Another potentially confusing issue is that the control variable and the limit variable are often the same. For example, during infant ventilation with a pulse (ie, rectangular) pressure waveform, pressure is both the control variable and the variable whose peak value is limited during inspiration. For adult ventilation with a pulse flow waveform, flow is also both the control and limit variable. However, it is a mistake to assume that the control and limit variables are always identical and that the words may be used interchangeably. It is possible to have a control variable but no limit variable. For example, if the control variable is flow with an ascending ramp waveform and there is no inspiratory pause, then there is no limit variable. The reason is that the set peak flow and tidal volume occur exactly at end-inspiration. Recall that the limit variable attains its peak value *before* end-inspiration. On the other hand, it is possible to have a limit variable but no control variable. For example, during ventilation with the proportional assist mode,[6] the waveforms for pressure, volume, and flow are not predetermined by the ventilator nor is inspiratory time preset. Therefore, by definition, there is no control variable. Yet both volume and flow reach preset maximum values before end-inspiration. In this case, however, the preset maximum values are *percentages* of the patient's spontaneously generated volume and flow, rather than fixed values. Therefore, we must conclude that the terms control variable and limit variable play unique and useful roles in the overall classification scheme.

In the equation of motion, pressure, volume, and flow are measured relative to their baseline values. Therefore, to maintain consistency, pressure, volume, and flow limits must be specified relative to their end-expiratory values. This is obvious for volume and flow—their values are zero at end-expiration. However, there is currently confusion about pressure limits. For example, in the pressure support mode, the pressure limit is specified as a value above end-expiratory pressure (ie, above PEEP, but the pressure limit on an infant ventilator is measured relative to atmospheric pressure. Again, to maintain consistency, *pressure limits must always be measured relative to the preset end-expiratory pressure.* It is the pressure change above end-expiratory pressure that determines the tidal volume. Thinking of the pressure limit as being measured above atmospheric pressure (and using a ventilator that controls it as such) rather than above end-expiratory pressure leads to clinical misjudgments. For example, a common mistake made by clinicians inexperienced in infant ventilation is to increase the PEEP without increasing the peak inspiratory pressure limit; then they wonder why the P_{CO_2} increases.

Cycle Variable—Inspiration always ends (ie, is cycled off) because some variable has reached a preset value. The variable that is measured and used to terminate inspiration is called the *cycle variable.* Deciding which variable terminates inspiration in a given ventilator can be confusing. For a variable to be used as a feedback signal (in this case a cycling signal) it must first be measured. Most third-generation adult ventilators allow the operator to set a tidal volume and inspiratory flowrate, which would lead one to believe that the ventilator could be volume-cycled. However, closer inspection reveals that these ventilators do not measure volume (which is consistent with the fact that all third-generation ventilators to date are flow-controllers). Rather, they set the inspiratory time necessary to achieve the set tidal volume with the set inspiratory flowrate, making them time-cycled. The tidal volume dial can be thought of as an inspiratory time dial calibrated in units of volume rather than time.

Some authors have asserted that a ventilator can have mixed cycling, which is contrary to the idea presented here that a ventilator can only control one variable at a time. The most common example given by these authors is a ventilator drive mechanism composed of a piston connected to a rod and a rotating crank. It is argued that one cannot distinguish between time (ie, inspiratory time set by the frequency at which the crank rotates) and volume (ie, the stroke volume of the piston) as the cycling variable.

This problem can be addressed as follows: First of all, such a mechanism has been used in two general applications. One is as the drive mechanism of a high frequency oscillator. In this application, for a given frequency and stroke volume of the piston, the pressure, volume, and flow delivered to the patient are usually the result of the relative impedances of the patient's respiratory system and the expiratory limb of the delivery circuit (usually configured as a variable low-pass filter). Here the pressure, volume, and flow waveforms delivered to the airway opening vary as respiratory system mechanics change so that the ventilator is a time-controller, as shown in Figure 6. Furthermore, it follows that inspiration does not end because a set pressure, or volume, or flow has been achieved, leaving time as the only reasonable cycling variable. The same argument can be applied to a system in which a piston is used to compress a reservoir bag.

The second application is exemplified by ventilators like those of the Emerson series or the Bourns LS-104. With these ventilators, if the inspiratory time is short enough and tidal volume large enough, then as the load imposed by a simulated patient increases, a point comes when the motor is overloaded and cannot meet the desired settings. In this case, it is clear that inspiratory time is lengthened for as long as it takes the piston to reach the end of its stroke. Thus, time is sacrificed for volume, and we must conclude that volume is the cycling variable.

Baseline Variable—The significant characteristic of expiration is how the ventilator affects the way the control variables return to their baseline values. The variable that is controlled during the expiratory time is the *baseline variable.* Note that in the equation of motion, pressure, volume, and flow are measured relative to end-expiratory or baseline values and are thus initially all zero. Although the baseline value of any of these variables could theoretically be controlled, pressure control is the most practical and is implemented by all commonly used ventilators.

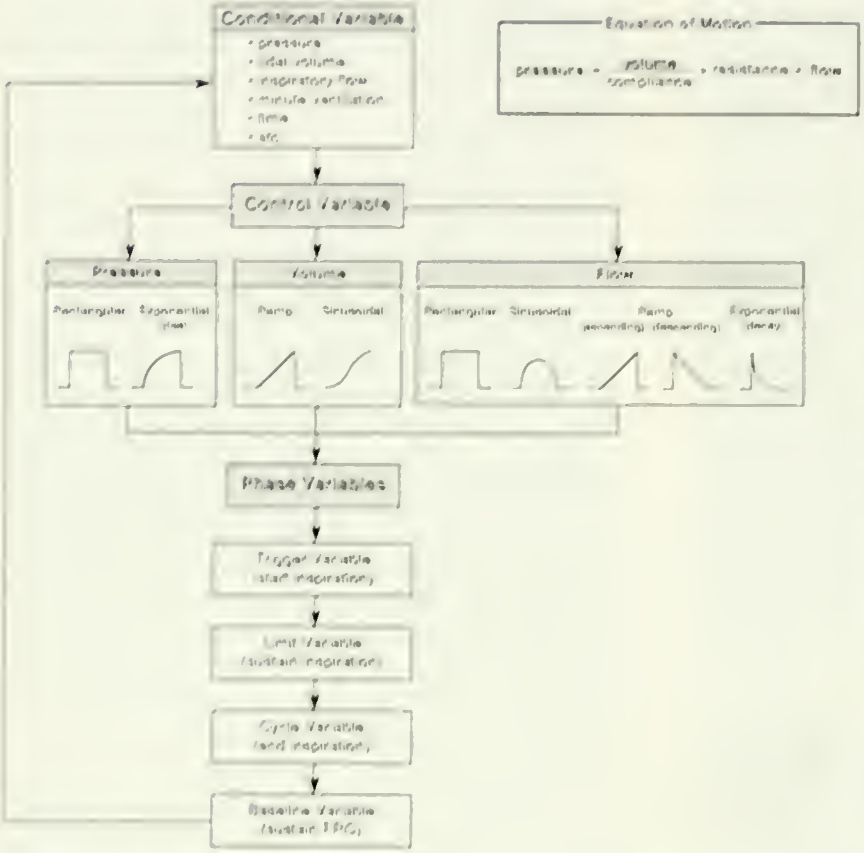

Fig. 6. This figure illustrates a new paradigm for understanding mechanical ventilators. It is based on a mathematical model known as the "equation of motion" for the respiratory system. This model indicates that during inspiration, the ventilator is able to directly control one and only one variable at a time (ie, pressure, volume, or flow). Some common waveforms provided by current ventilators are shown for each control variable. Pressure, volume, flow, and time are also used as phase variables that determine the parameters of each ventilatory cycle (eg, trigger sensitivity, peak inspiratory flowrate or pressure, inspiratory time, and baseline pressure). Modes of ventilation are simply jargon for various combinations of control and phase variables. The diagram is drawn as a flow chart to emphasize that each breath may have a different set of control and phase variables depending on the mode of ventilation desired.

The ability of a ventilator to control the baseline variable means, for practical purposes, the ability to control expiratory transrespiratory pressure. Notice that the emphasis is on *transrespiratory* pressure rather than *airway* pressure. This distinction is made for two reasons. First, it is to accommodate the situation in which a negative pressure ventilator is able to maintain a negative body surface pressure during expiration. The baseline transrespiratory pressure difference (airway pressure minus body surface pressure) in this case is positive, just as when conventional end expiratory pressure is used. However, end expiratory airway pressure is zero. Thus, thinking of end-expiratory pressure in terms of airway pressure rather than transrespiratory pressure is misleading.

The second reason for the distinction between transrespiratory and airway pressures is that it might be desirable to apply a negative transrespiratory pressure change to facilitate expiration in the face of increased airway resistance. Schulze et al have described this technique as "resistive unloading" or "negative ventilator resistance." This is similar to the concept of using pressure support to decrease the resistive work of inspiration, except that the change in transrespiratory pressure is negative instead of positive, and an expiratory pressure limit is set below the baseline rather than above it. More precisely, we set one baseline to assist expiratory flow and then another when expiratory flow ceases, in order to restore functional residual capacity. This prevents gas trapping without the risk of alveolar collapse associated with the old style negative end-expiratory pressure (NEEP).

We call the first baseline setting transient expiratory assist pressure (TEAP) and the second end-expiratory pressure (EEP). If the ventilatory frequency is set to zero, we can say that the patient is on a constant baseline pressure or constant airway pressure (CAP).

Conditional Variables

For each breath, the ventilator must choose a specific pattern of control and phase variables. The ventilator may either keep this pattern constant for each breath or it may introduce other patterns (eg, one for mandatory and one for spontaneous breaths). Some ventilators are capable of complex patterns such as two types of mandatory breaths (one normal, one sigh) and two types of spontaneous breaths (eg, with two different pressure limits). In essence, the ventilator must 'decide' which pattern of control and phase variables to implement before each breath, depending on the value of some preset *conditional variables*. A simple example would be the Bennett MA-1 in the control mode. Each breath is time-triggered, flow-limited, and volume-cycled. The trigger, flow, and volume variables have preset values (eg, frequency = 20 cycles/min, inspiratory flow = 60 L/min, and tidal volume = 750 mL). However, every few minutes a sigh breath is introduced with a different set of phase variable values (eg, frequency = 2 sighs every 15 minutes, tidal volume = 1,500 mL).

How did the ventilator 'know' to do this? Conceptually, we can say that before each breath pattern is selected, the ventilator implements a control logic algorithm made up of if-then statements). It examines the value of some conditional variable to see if a preset threshold value has been reached. If the threshold has been met, then one pattern is selected, if not, then another pattern is selected. In the case of the MA-1, the conditional variable is time—once a preset time interval has elapsed, the ventilator switches to the sigh pattern.

Another example is the SIMV (synchronized intermittent mandatory ventilation) mode. If the ventilator detects a patient effort and the SIMV window has not closed, then a spontaneous breath is delivered. If an effort is not sensed and the window has closed, then a mandatory breath is delivered. Here there are two conditional variables, pressure (or flow) and time.

Yet another example is the mandatory minute ventilation mode. Here, the conditional variable is exhaled minute ventilation. When minute ventilation falls below a preset threshold, the ventilator may switch from a spontaneous breath pattern to a mandatory pattern (eg, the Bear 5) or it may change the parameters of the spontaneous breath (eg, the Hamilton Veolar increases the pressure limit). In the future, ventilators may be able to switch patterns based on arterial blood gas values. Several conditional variables in different combinations will produce a large variety of breath patterns within each mode.

Figure 6 illustrates the entire ventilator classification scheme in terms of an algorithm describing the events that take place from breath to breath in the ventilator's control system.

Output Waveforms

Just as the study of cardiac physiology involves the study of electrocardiograms and blood pressure waveforms, the study of ventilator operation requires the examination of output waveforms. The waveforms of interest, of course, are the pressure, volume, and flow waveforms used throughout this discussion.

Output waveforms are graphed in groups of three (Fig. 7). The conventional order of presentation is pressure, volume, and flow. This order is based on the mathematical convention used for the equation of motion, a specific example of a general class of expressions called first-order linear differential equations. Convention also dictates that positive values (above the horizontal axis) correspond to inspiration and negative values (below the horizontal axis) correspond to expiration. The horizontal axes of all three graphs are the same and have the units of time. The vertical axes are in units of the measured variables (eg, cm H_2O for pressure). For the purpose of identifying output waveforms, the specific baseline values of each variable are irrelevant. Therefore, the origin of the vertical axis is labeled zero. What is important is the relative magnitude of each of the variables and how the value of one affects or is affected by the value of the others.

Remember that the waveforms used to define categories of ventilator output are idealized. That is, they are precisely defined by mathematical equations and are meant to characterize the operation of the ventilator's control system. As such, they do not show the minor deviations or 'noise' often seen in waveforms recorded during actual ventilator use. These waveform imperfections can be caused by a variety of factors such as vibration and turbulence. Also, the appearance of the waveform is affected by the scaling of the time axes

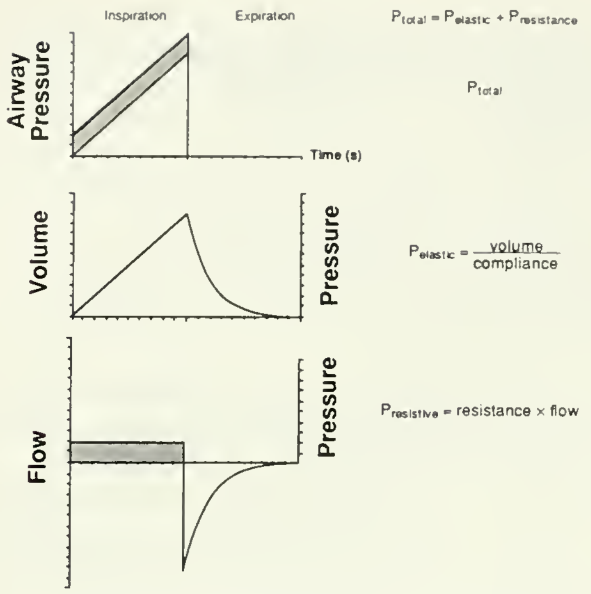

Fig. 7. This figure illustrates some conventions for the presentation of graphic data. It shows the theoretical output waveforms for flow-controlled inspiration with a pulse (ie, rectangular) flow waveform. The order of presentation is pressure, volume, and flow, according to the order specified by the equation of motion. Note that the volume waveform is the same shape as the transthoracic or lung pressure waveform (ie, pressure due to elastic recoil). The flow waveform is the same shape as the transairway pressure waveform (ie, pressure due to airway resistance). If all the pressure scales are the same, then the height of the airway pressure waveform at any instant is the sum of the heights of the other two waveforms as indicated by the shaded and unshaded portions of the figure. The origin of the airway pressure waveform is the end-expiratory pressure; the origins of the volume and flow waveforms are both zero.

No ventilator is an ideal controller, and ventilators are designed to only approximate a particular waveform. Idealized or standard waveforms are nevertheless helpful because they are common in other fields (eg, electrical engineering), which makes it possible to use mathematical procedures and terminology that have already been developed. For example, a standard mathematical equation may be used to describe the most common waveforms for each control variable. This known equation may be substituted into the equation of motion, which is then solved to obtain the equations of the other two variables. Once the equations for pressure, volume, and flow are known, they are easily graphed. This is the process used to generate the graphs in Figure 7.

Ventilator-Alarm Systems

Like many things, alarms have increased in number and complexity. Fortunately, the system we have been using to classify ventilators is easily adaptable for the functional classification of alarms. Day and MacIntyre[13] have stressed that the goal of ventilator alarms is to warn of events. They define an event as any condition or occurrence that requires clinician awareness or action. *Technical events* are those involving an inadvertent change in the ventilator's performance; *patient events* are those involving a change in the patient's clinical status that can be detected by the ventilator.[13] A ventilator may be equipped with any conceivable vital sign monitor, so we need to specify the appropriate scope of surveillance. The most logical scope would include the ventilator's mechanical/electronic operation, and those variables associated with the mechanics of breathing (ie, pressure, volume, flow, and time). Because the ventilator is in intimate contact with exhaled gas, it seems appropriate to include the analysis of exhaled oxygen and carbon dioxide concentrations as possible variables to monitor. Other devices, such as hemodynamic and cardiac monitors, and pulse oximeters seem less appropriate for inclusion in ventilator design.

Alarms may be audible, visual, or both, depending upon the seriousness of the alarm condition. Visual alarms may be as simple as colored lights or may be as complex as alphanumeric messages to the operator indicating the exact nature of the fault condition. Specifications for an alarm event should include (1) conditions that trigger the alarm; (2) the alarm response in the form of audible and/or visual messages; (3) any associated ventilator response such as termination of inspiration or failure to operate; and (4) an indication that the alarm must be manually reset or resets itself when the alarm condition is rectified.

Input Power Alarms: ***Electric Power Loss***—Most ventilators have some sort of battery backup in the case of electrical power failure, even if the batteries only power alarms. Ventilators typically have alarms that are activated if the electrical power is cut off while the machine is still switched on (eg, if the power cord is accidentally pulled out of the wall socket). If the ventilator is designed to operate on battery power (eg, transport ventilators), an alarm is usually present to warn of low-battery conditions.

Input Power Alarms: ***Pneumatic Power Loss***—Ventilators that use pneumatic power have alarms that are activated if either the oxygen or air supply is cut off or reduced below some specified driving pressure. In some cases, the alarm is activated by an electronic pressure switch (eg, Bennett 7200) but in others, the alarm is pneumatically operated as a part of the blender (eg, Siemens Servo 900C).

Control Circuit Alarms—Control circuit alarms are those that either warn the operator that the set control-variable parameters are incompatible (eg, inverse I-E ratio) or indicate that some aspect of a ventilator self-test has failed. In the latter case, there may something wrong with the ventilator control circuitry itself (eg, a microprocessor failure) and the ventilator generally responds with some generic message like Ventilator Inoperative.

Output Alarms—Output alarms are those that are triggered by an unacceptable state of the ventilator's output. More specifically, an output alarm is activated when the value of a control variable (pressure, volume, flow, or time) falls outside an expected range. Some possibilities include

Output Alarms—*Pressure*: Pressure alarms may be available for the following conditions:

> *High and low peak airway pressure* indicating a possible endotracheal tube obstruction or leak in the patient circuit, respectively.
>
> *High and low mean airway pressure* indicating a possible leak in the patient circuit or a change in ventilatory pattern that might lead to a change in the patient's oxygenation status (ie, within reasonable limits, oxygenation is roughly proportional to mean airway pressure).
>
> *High and low baseline pressure (PEEP or CPAP)* indicating a possible patient-circuit or exhalation-manifold obstruction (or inadvertent PEEP) and disconnection of the patient from the patient circuit, respectively.
>
> *Failure of airway pressure to return to baseline within a specified period* indicating a possible patient-circuit obstruction or exhalation-manifold malfunction.

Output Alarms: ***Volume***—*High and low exhaled tidal volume* indicating changes in respiratory system time constant during pressure-controlled ventilation or possible disconnection of the patient from the patient circuit.

Output Alarms: ***Flow***—*High and low exhaled minute ventilation* indicating hyperventilation (or possible machine self-triggering) and possible apnea or disconnection of the patient from the patient circuit, respectively.

Output Alarms—*Time*: Time alarms may be available for the following conditions.

> *High or low ventilatory frequency* indicating hyperventilation (or possible machine self-triggering) and possible apnea, respectively.
>
> *Inspiratory time too long or too short:* Inspiratory time too long indicates a possible patient circuit obstruction or exhalation manifold malfunction. Inspiratory time too short indicates that adequate tidal volume may not be delivered (in a pressure-controlled mode) or that gas distribution in the lungs may not be optimal.
>
> *Expiratory time too long or too short:* Expiratory time too long may indicate apnea. Expiratory time too short may warn of alveolar gas trapping (ie, expiratory time should be ≥ five time constants of the respiratory system).

Output Alarms—*Inspired Gas*: Inspired gas alarms may be available for *high/low inspired gas temperature* and *high/low* F_{IO_2}.

Output Alarms—*Expired Gas*: Because ventilators are designed to control the mechanical results of exhalation, they may be easily adapted to the analysis of exhaled gas composition and alarms may be set for specific parameters.

Exhaled Carbon Dioxide Tension: End tidal carbon dioxide monitoring may reflect arterial carbon dioxide tension and thus indicate the level of ventilation. Mean exhaled carbon dioxide tension and minute ventilation measurements could provide information about carbon dioxide production and allow calculation of the respiratory exchange ratio and dead-space-to-tidal-volume ratio.

Exhaled Oxygen Tension:[14] Analysis of end-tidal and mean exhaled oxygen tension may provide information about gas exchange and could be used with CO_2 data to calculate the respiratory exchange ratio.

How To Speak Machine Language

A successful classification scheme should easily accommodate the differing needs and interests of its intended audience. An administrator may need to know only where and for what type of patient the ventilator can be used (making the input power, and control variables important). In addition to this information, the clinician wants to know how the ventilator is operated (making phase variables and output characteristics of interest). The student, educator, or researcher may need to understand the internal workings of the ventilator (requiring a knowledge of drive mechanism, compressor motor/linkage, output control valves, and control variables/waveforms). Certainly, the manufacturer's representatives should be conversant with all of this information and be able to provide it to interested parties. The classification system presented here is designed to meet these needs in a logical, consistent, and unambiguous manner. Let us explore some ways that it can be used to communicate ideas in ventilator lingo.

The average clinician needs to describe very little about a ventilator during the course of the daily routine. Hence, the terms volume ventilator and pressure ventilator are often heard, referring to ventilators that allow a preset inspiratory pressure or tidal volume, respectively. Confusion quickly arises, however, when one attempts to describe how a ventilator operates, especially when speaking to someone who has little experience with the subject. Terms like pressure generator or flow generator are often used but may be ambiguous because all ventilators generate pressure, volume, and flow all at once. The idea one would like to convey is that a ventilator controls a particular variable; hence, the terms pressure-controller, volume-controller, and flow-controller are more specific.

The outline approach to classification lends itself to varying degrees of detail. For example, a simple description of an Emerson Iron Lung is an electrically powered negative-pressure-controller that produces a sinusoidal pressure waveform. A more detailed description adds that it is time-triggered, time-cycled, provides no baseline pressure control, and uses an electrically controlled drive mechanism consisting of an diaphragm, rotating crank, and connecting rod driven by an electric motor. It operates in the control mode only and has no alarms.

A simple description of a more complex ventilator like the Bennett 7200 is an electrically powered pressure- or flow-controller. A more detailed description includes time-, pressure-, flow-, or manually-triggered; pressure- or volume-limited; time-, volume-, or flow-cycled; controls baseline pressure (PEEP/CPAP); and uses an electronic (microprocessor) control circuit and a drive mechanism/output control valve consisting of a pressure regulator and proportional valve with a pneumatic diaphragm exhalation manifold valve. One can go on and list the multitude of operating modes and alarms. Needless to say, the more complicated the ventilator, the more involved the description.

Perhaps more commonly, clinicians need to refer to a mode of ventilation rather than to a specific ventilator per se. The most general terms seem to be volume-controlled versus pressure-controlled ventilation. The term pressure-controlled ventilation should cause no problems but the use of volume-controlled ventilation may be confusing. A strict interpretation of the classification system presented in this discussion would require a distinction between volume-controlled and flow-controlled modes. However, in the interest of convenience, the term volume-controlled should be acceptable for both. Any breath that is directly flow-controlled is indirectly volume-controlled, and vice versa. (But, when describing a specific ventilator rather than a mode of ventilation, you should make the distinction between volume and flow control.) More information about the mode of ventilation can be conveyed by stating the control and phase variables for mandatory and spontaneous breaths. In fact, you can communicate most of the important information by just specifying the phase variables for the mandatory and spontaneous breaths. For example, a description of SIMV with pressure support might be that mandatory breaths are time-or pressure-triggered, flow-limited, and time-cycled; spontaneous breaths are pressure-triggered, pressure-limited, and flow-cycled. What more do you need?

Use of this system may seem uncomfortable at first, especially to those who are already accustomed to some other system. Yet, if the basic ideas are learned, the rest is easy. It is similar to the game of checkers—only a few simple rules but an almost infinite variety of playing strategies and tactics. But if two people playing the game do not know and use the same set of rules, confusion and conflict result. To carry the checker-game analogy further, the person who sees the similarity of positional patterns is much more likely to be successful at the game than the player who sees each position as different and unique. In the same way, the clinician who sees the similarity of design features and applies the same operational definitions to all ventilators more easily understands ventilators and modes of ventilation than does the clinician who tries to memorize endless lists of different features.

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Technical Description and Classification of Modes of Ventilator Operation

Richard D Branson RRT and Robert L Chatburn RRT

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Mr Branson is Clinical Instructor, Division of Trauma/Critical Care, Department of Surgery, University of Cincinnati College of Medicine, University Hospital, Cincinnati, Ohio, and Mr Chatburn is Director, Pediatric Respiratory Care, Rainbow Babies and Childrens Hospital, University Hospitals of Cleveland, Cleveland, Ohio.

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Reprints: Richard D Branson RRT, Department of Surgery ML-558, Univ of Cincinnati Medical Center, 231 Bethesda Ave, Cincinnati OH 45267-0558.

Introduction

Mechanical ventilation is frequently required to support patients in respiratory failure. Although the goals of mechanical ventilation are fairly consistent, a variety of techniques and a seemingly never-ending array of devices are applied to attain those goals. In this regard, mechanical ventilation is much more a practiced art than a science.

Nowhere is this more evident than in the clinician's choice of mode of operating the ventilator. Each mode has its staunch supporters and its equally determined detractors. No group seems to understand the approach of another and most fail to realize that experience and skill with a specific mode are probably the single greatest determinants of success with that mode. Further, the number of available operational modes has tripled in the past 12 years, and considerable confusion exists regarding labeling and function.

In this paper we review the currently available modes, their terminology, and their function in light of the classification system proposed by Chatburn.[1] Following these descriptions, we present evidence that suggests that considerable repetition exists in names and descriptions of operational modes. When repetition and overlap are eliminated, the proliferation of names becomes unnecessary—only two mutually exclusive modes remain.

Concepts

According to Chatburn, a mode can be classified based upon control, phase, and conditional variables.[1]

Control Variables

A control variable is the variable (ie, pressure, volume, flow, or time) that the ventilator manipulates to cause inspiration. A control variable is identified by the fact that its behavior remains constant despite changes in ventilatory load (ie, despite changes in compliance and resistance of the patient or model, the ventilator 'sacrifices' all other preset variables to keep the control variable constant).

Phase Variables

Phase variables are variables (eg, pressure, volume, flow, and time) that are measured and used to initiate some phase of the ventilatory cycle. These include trigger, limit, and cycle.

Trigger—The trigger variable causes inspiration to begin. For instance, when the patient creates an inspiratory effort, causing pressure in the circuit to fall below the sensitivity setting, inspiration is said to be pressure-triggered. Of course, inspiration can be flow- or volume-triggered as well. In any of these three cases, we can also say that the mode is patient-triggered. If inspiration is time-triggered, then we can say that inspiration is machine-triggered. The distinction between patient-triggered and machine-triggered is a simpler and perhaps more easily used description. However, understanding the variable used to trigger inspiration is necessary for a complete understanding of how a particular ventilator operates in the specified mode. For instance, the Puritan-Bennett 7200ae can operate in what we currently call the synchronized intermittent mandatory ventilation (SIMV) mode, using either flow- or pressure-triggering of spontaneous (patient-triggered) breaths.[2] The variable used for triggering does not change the mode of operation, it remains SIMV.

Limit—The limit variable is the variable (pressure, volume, or flow) with a preset maximum value during an assisted inspiration. When the limit variable is met, inspiration is not terminated. For instance, during pressure support ventilation (PSV), a pressure limit is selected, but even after this level is met, inspiration continues until attainment of a predetermined flowrate or percentage of initial flowrate causes inspiration to be terminated.

Cycle—The cycle variable, when reached, terminates inspiration. During PSV, inspiration is flow-cycled when the inspiratory flowrate decays to a preset minimum flow or percentage of initial flowrate. Previous classification systems have used the term cycle to describe both initiation and termination of inspiration (ie, "cycle on" and "cycle off"). This can be confusing and leads to circumstances in which a mode is described as both patient- and volume-cycled.

Conditional Variables

Conditional variables are those variables that, alone or in combination, are examined by the ventilator's control logic. The status of the conditional

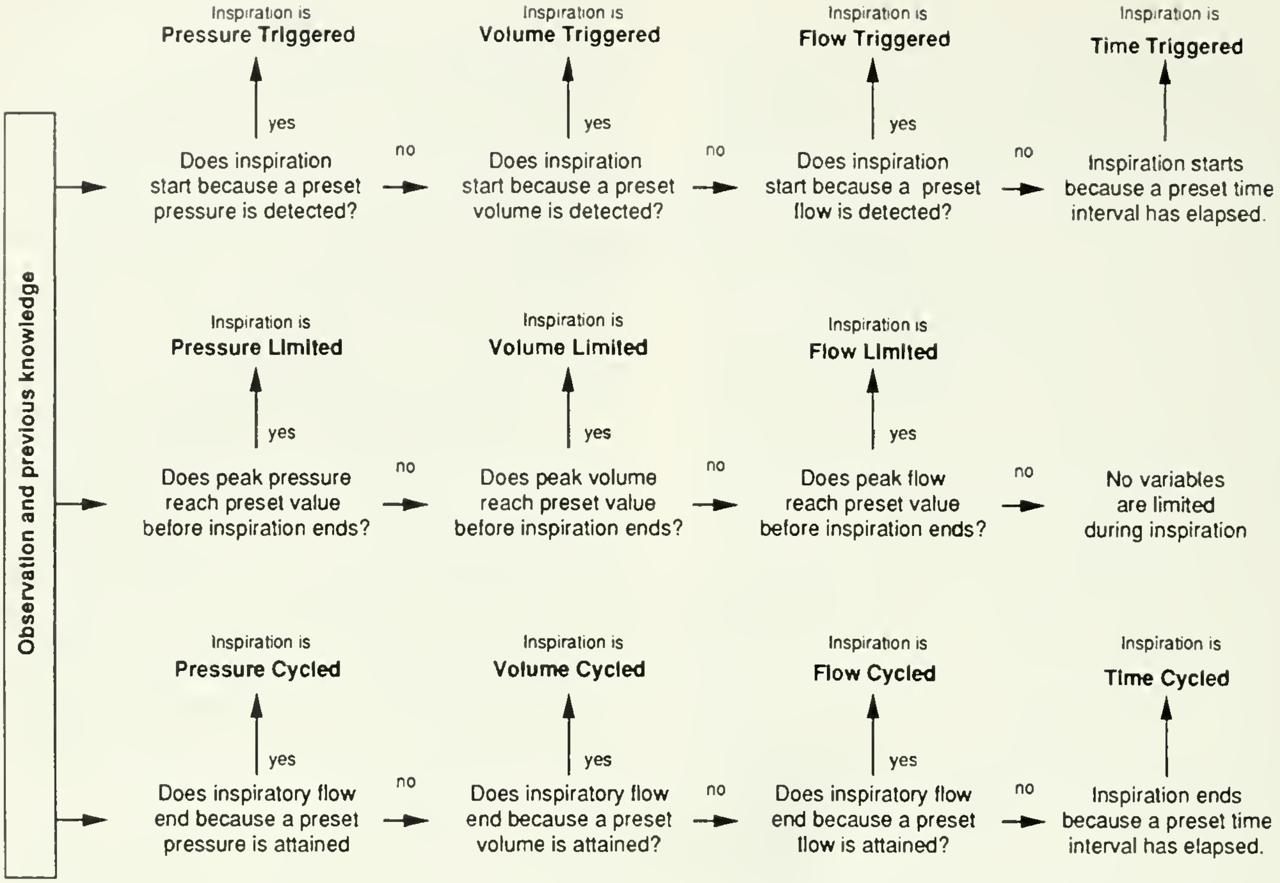
Observation and previous knowledge
Inspiration is Pressure Triggered
yes
Does inspiration start because a preset pressure is detected?
no
Inspiration is Volume Triggered
yes
Does inspiration start because a preset volume is detected?
no
Inspiration is Flow Triggered
yes
Does inspiration start because a preset flow is detected?
no
Inspiration is Time Triggered
Inspiration starts because a preset time interval has elapsed.
Inspiration is Pressure Limited
yes
Does peak pressure reach preset value before inspiration ends?
no
Inspiration is Volume Limited
yes
Does peak volume reach preset value before inspiration ends?
no
Inspiration is Flow Limited
yes
Does peak flow reach preset value before inspiration ends?
no
No variables are limited during inspiration
Inspiration is Pressure Cycled
yes
Does inspiratory flow end because a preset pressure is attained
no
Inspiration is Volume Cycled
yes
Does inspiratory flow end because a preset volume is attained?
no
Inspiration is Flow Cycled
yes
Does inspiratory flow end because a preset flow is attained?
no
Inspiration is Time Cycled
Inspiration ends because a preset time interval has elapsed.

Fig. 1. Criteria for determining the phase variables during a ventilator-supported breath. (Reprinted from Reference 1.)

variable determines which of two or more breath types is delivered. For instance, in the mandatory minute ventilation mode (MMV) of the Bear 5 ventilator, PSV can be provided; but if a set minute ventilation ($\dot{V}_E$) is not reached, a mandatory breath is delivered.[3] In this case, $\dot{V}_E$ is the conditional variable that determines the type of breath to be delivered, pressure-supported or mandatory. Conditional variables are also important in the SIMV mode, in which (based on a timing window and patient inspiratory effort) the ventilator delivers either a pressure-triggered (patient) or time-triggered (machine) breath. In this instance, time and patient effort are the conditional variables used to determine triggering.

A mode, then, according to this scheme, is a specific combination of control, phase, and conditional variables defined for both mandatory and spontaneous breaths.[1] An algorithm for determining trigger, limit, and cycle variables is shown in Figure 1.

Mandatory vs Spontaneous Breaths

Another important concept is differentiation between mandatory and spontaneous breaths.

Mandatory Breath—a machine-supported breath that is either initiated or terminated by the ventilator rather than by the patient's physiology or ventilatory drive. This means that any breath that is time-triggered and/or time-cycled is a mandatory breath.

Spontaneous Breath—either an unsupported breath or a machine-supported breath that is both patient-initiated and patient-terminated.

Understanding this differentiation is critical to further discussion of specific modes. This differentiation suggests that all breaths in the PSV mode are spontaneous, whereas breaths triggered by the patient in the assist/control mode are man-

Table 1. Expanded Classification of Modes of Ventilator Operation

	Mandatory Breath				Spontaneous Breath					Control Logic	
Mode	Control	Trigger	Limit	Cycle	Control	Trigger	Limit	Cycle	Supported	Conditional Variable	Action
CMV*	Pressure, volume, or flow	Time	Pressure, volume, or flow	Time, pressure, volume, or flow	—	—	—	—	—	—	—
A/C	Pressure, volume, or flow	Time, pressure, volume, or flow	Pressure, volume, or flow	Time, pressure, volume, or flow	—	—	—	—	—	Time or patient effort	Machine-to-patient triggered
AMV	Pressure, volume, or flow	Pressure, volume, or flow	Pressure, volume, or flow	Time, pressure, volume, or flow	—	—	—	—	—	—	—
IMV	Pressure, volume, or flow	Time	Pressure, volume, or flow	Time, pressure, volume, or flow	Pressure	Pressure, volume, or flow	Pressure	Pressure	No	—	—
SIMV	Pressure, volume, or flow	Time, pressure, volume, or flow	Pressure, volume, or flow	Time, pressure, volume, or flow	Pressure	Pressure, volume, or flow	Pressure	Pressure	No	Time or patient effort	Machine-to-patient triggered
CPAP	—	—	—	—	Pressure	Pressure, volume, or flow	Pressure	Pressure	No	—	—
PCV	Pressure	Time	Pressure	Time	—	—	—	—	—	—	—
PC-IMV	Pressure	Time	Pressure	Time	Pressure	Pressure, volume, or flow	Pressure	Pressure	No		
PC-SIMV	Pressure	Time, pressure, volume, or flow	Pressure	Time	Pressure	Pressure, volume, or flow	Pressure	Pressure	No	Time or patient effort	Machine-to-patient triggered
PCIRV	Pressure	Time	Pressure	Time	Pressure						
APRV	Pressure	Time or pressure	Pressure	Time	Pressure	Pressure, volume, or flow	Pressure	Pressure	No	Time or patient effort	Machine-to-patient triggered
PSV	—	—	—	—	Pressure	Pressure, volume, or flow	Pressure	Volume	Yes		
MMV	Volume or flow	Time	Volume or flow	Time, volume, or flow	Pressure	Pressure, volume, or flow	Pressure	Pressure or volume	Yes*	Minute ventilation, time	Spontaneous to-mandatory breath
VAPS	—	—	—	—	Pressure, volume, or flow	Pressure, volume, or flow	Pressure	Pressure, volume, or flow	Yes*	Tidal volume	Pressure-to-volume control
BiPAP	Pressure	Time	Pressure	Time	Pressure	Pressure	Pressure	Pressure	No	—	—

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*CMV = continuous mandatory ventilation; NA = not applicable; A/C = assist/control; AMV = assisted mechanical ventilation; IMV = intermittent mandatory ventilation; SIMV = synchronized mandatory ventilation; CPAP = continuous positive airway pressure; PCV = pressure-controlled ventilation; PC-IMV = pressure-controlled IMV; PCIRV = PC inverse-ratio ventilation; APRV = airway pressure release ventilation; PSV = pressure support ventilation; MMV = mandatory minute ventilation; VAPS = volume-assisted pressure support; BiPAP = bilevel positive airway pressure.

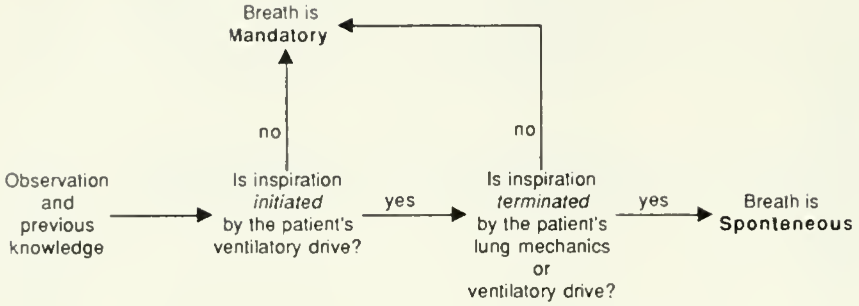
Breath is Mandatory
no
no
Observation and previous knowledge
Is inspiration *initiated* by the patient's ventilatory drive?
yes
Is inspiration *terminated* by the patient's lung mechanics or ventilatory drive?
yes
Breath is Sponteneous

Fig. 2. Algorithm defining spontaneous and mandatory breaths. If the breath is triggered according to a preset frequency or minute ventilation or cycled according to a preset frequency or tidal volume, the breath is mandatory. All other breaths are spontaneous.

datory. A spontaneous breath can be one of two types. Unsupported spontaneous breaths are those to which the ventilator does not add positive pressure (eg, during CPAP). Supported spontaneous breaths are breaths that, although initiated and terminated by the patient, receive positive pressure from the ventilator to increase their volume (eg, during PSV). Figure 2 depicts the algorithm for determining mandatory vs spontaneous breaths.

Analysis of Current Ventilator Modes

Controlled Mechanical Ventilation (CMV)

Definition—Controlled mechanical ventilation is a mode of ventilator operation in which all breaths are delivered by the ventilator at a preset frequency (f), volume or pressure, and inspiratory flowrate ($\dot{V}$). Patient-triggering, via any variable, is not possible.

Other Terms—The CMV abbreviation has been used for continuous mechanical ventilation, continuous mandatory ventilation, controlled mechanical ventilation, and controlled mandatory ventilation.[4-7] Interestingly, authors have preserved the abbreviation while choosing its meaning haphazardly. CMV is also frequently called volume-controlled ventilation (VCV) or just simply control mode (no abbreviation).[4,5]

Manufacturers' Terms—Currently available mechanical ventilators refer to CMV as CMV Assist-Control, Control, Volume-Control, and a host of others. In some cases, this mode strictly adheres to the above definition, but in others the patient is allowed to trigger mandatory breaths by exceeding the flow-, volume-, or pressure-sensitivity setting. This mode is often called assist/control. On many ventilators, CMV and assist/control are the same, the only difference being the sensitivity setting. For instance, with the Hamilton Veolar, if the CMV mode is desired, the sensitivity control is dialed to its least sensitive position (–20 cm H_2O).[8] Otherwise, patient-triggering is possible. (Of course, it would be wise to sedate and paralyze the patient in this situation.)

Chatburn's Classification—Using the schema described by Chatburn, CMV is classified as volume-/flow- or pressure-controlled; time-triggered; volume-, pressure-, or flow-limited; and volume-, pressure-, flow-, or time-cycled (Table 1). All breaths are mandatory breaths (Fig. 3). Simplifying this by substituting the more generic terms, CMV is pressure or volume-flow controlled; machine-triggered; and machine-cycled (Table 2). It should be obvious to the reader at this point that conveying the message that the patient is "on CMV" fails to describe the mode of operation. Depending on the ventilator used and local practice, CMV could mean that mandatory breaths are pressure- or

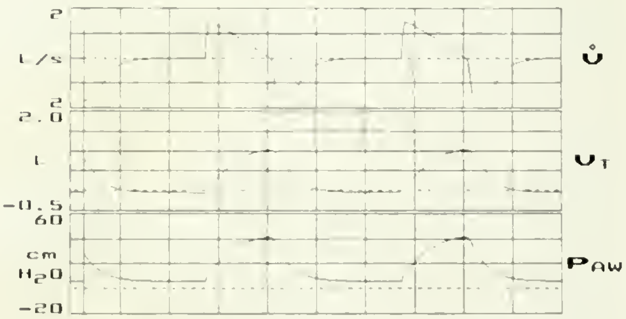

Fig. 3. Flow, volume, and pressure waveforms for what is commonly referred to as CMV. The suggested term for this mode is volume-controlled, continuous mandatory ventilation, machine-triggered.

Table 2. Simplified Classification of Modes of Ventilator Operation

	Mandatory Breath			Spontaneous Breath	Control Logic	
Mode	Control	Trigger	Cycle	Supported	Conditional Variable	Action
CMV*	Pressure, volume, or flow	Machine	Machine	NA	—	—
A/C	Pressure, volume, or flow	Machine or Patient	Machine	NA	Time or patient effort	Machine-to-patient triggered
AMV	Pressure, volume, or flow	Patient	Machine	NA	—	—
IMV	Pressure, volume, or flow	Machine	Machine	No	—	—
SIMV	Pressure, volume, or flow	Machine or patient	Machine	No	Time or patient effort	Machine-to-patient triggered
CPAP	—	—	—	No	—	—
PCV	Pressure	Machine	Machine	NA	—	—
PC-IMV	Pressure	Machine	Machine	No		
PC-SIMV	Pressure	Machine or patient	Machine	No	Time or patient effort	Machine-to-patient triggered
PCIRV	Pressure	Machine	Machine	NA		
APRV	Pressure	Machine	Machine	No		
PSV	—	—	—	Yes		
MMV	Pressure, volume, or flow	—	Machine	Yes†	Minute ventilation and time	Spontaneous-to-mandatory breath
VAPS	—	—	—	Yes	Tidal volume	Pressure-to-volume control
BiPAP	Pressure	Machine	Machine	No	—	—

*CMV = continuous mandatory ventilation; NA = not applicable; A/C = assist/control; AMV = assisted mechanical ventilation; IMV = intermittent mandatory ventilation; SIMV = synchronized mandatory ventilation; CPAP = continuous positive airway pressure; PCV = pressure-controlled ventilation; PC-IMV = pressure-controlled IMV; PCIRV = PC inverse-ratio ventilation; APRV = airway pressure release ventilation; PSV = pressure support ventilation; MMV = mandatory minute ventilation; VAPS = volume-assisted pressure support; BiPAP = bilevel positive airway pressure.

† Optional.

volume-controlled; patient- (using any of the possible variables) and/or machine-triggered; pressure-, volume-, or flow-limited; and time-, flow-, volume-, or pressure-cycled. Of course, one could take consolation in the fact that, at least, the breaths are all machine-cycled. Thus, we believe that the term CMV, as currently used, is inadequate to describe the ventilator's operation.

Assist/Control Ventilation (A/C)

Definition—Assist/control ventilation (A/C) is a mode of ventilator operation in which mandatory breaths are delivered at a set f, pressure or volume, and inspiratory flow. Between machine-initiated breaths, the patient can trigger the ventilator and receive a mandatory breath at the volume or pressure set on the ventilator.[4-7] Machine- and patient-triggered breaths are delivered using the same limit and cycle variables.

Other Terms—Assist/control ventilation has been described in the literature as assisted mechanical ventilation (AMV), assisted ventilation, and CMV with assist.

Manufacturers' Terms—Many ventilators use the term CMV to describe assist/control, the only difference being the position of the sensitivity setting. Other terms include assist-control and volume control.

Chatburn's Classification—Regardless of the terminology used, A/C is described as pressure- or volume-controlled; time-, pressure-, flow-, or volume-triggered; pressure-, flow-, or volume-limited; and flow-, volume-, pressure-, or time-cycled (Table 1). Again, it is obvious that, although a group of clinicians with similar training understand what the term A/C indicates, the term is too imprecise to convey real meaning. Using the simplified version, A/C can be described as pressure- or volume-/flow-controlled; machine- and patient-triggered; and machine-cycled. All breaths are referred to as mandatory breaths (Fig. 4).

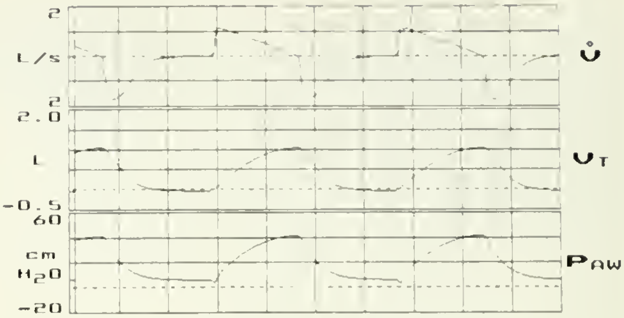

Fig. 4. Flow, volume, and pressure waveforms for what is commonly referred to as A/C. The difference between the CMV and A/C waveforms is in the negative pressure deflections prior to breath delivery. The suggested term for this mode is volume-controlled, continuous mandatory ventilation, combined trigger. Combined trigger means mandatory breaths can be both machine- and patient-triggered.

Assisted Mechanical Ventilation (AMV)

Definition—AMV is a proposed version of A/C in which there is no set frequency[4-7] (ie, all breaths are patient-triggered and delivered at the ventilator's set tidal volume or pressure).

Other Terms—The pure form of AMV, without a set backup rate, is not often used or discussed. The term assisted ventilation has been used but frequently alludes to A/C.

Manufacturers' Terms—To our knowledge, no manufacturer labels a mode of operation as only Assist. AMV could be produced by placing the patient in whatever version of A/C is available and setting the respiratory frequency at 0. This would be impossible on those ventilators with a minimum rate greater than 0 in the CMV or A/C mode.

Chatburn's Classification—True assist-mode ventilation is classified as volume-/flow- or pressure-controlled; pressure-, flow-, or volume-triggered; flow-, volume-, or pressure-limited; and time-, flow-, volume-, or pressure-cycled (Table 1). The proposed system (Table 2) classifies this mode as volume-/flow- or pressure-controlled, patient-triggered, and machine-cycled. Aside from not specifying whether volume or pressure is preset and, therefore, not specifying what limits inspiration, this describes the essence of assisted ventilation.

Intermittent Mandatory Ventilation (IMV)

Definition—Intermittent mandatory ventilation (IMV) is a mode of ventilator operation in which mandatory (machine) breaths are delivered at a set f and volume or pressure. Between machine breaths, the patient can breathe spontaneously from either a continuous flow of gas or a demand system.[4-7,9-14]

Other Terms—For the most part, IMV has survived interchangeable name calling (and even derogatory name-calling). At one time, IMV was frequently referred to as intermittent demand ventilation (IDV) and was occasionally called "intermittent respiratory failure" by its most ardent critics.[15]

Manufacturers' Terms—The terms IMV or SIMV (discussed later) are used by most manufacturers. The term IMV is sometimes linked with continuous positive airway pressure (CPAP) on the mode selection switch, dial, or key pad.

Chatburn's Classification—As a mode, IMV presents the new problem of classifying both mandatory and spontaneous breaths. According to Chatburn, mandatory breaths during IMV are volume-/flow- or pressure-controlled; time-triggered; pressure-, volume-, or flow-limited; and pressure-, volume-, flow-, or time-cycled. Spontaneous breaths are not controlled and therefore have no trigger, limit, or cycle variable if a continuous flow of gas is used. Demand systems (ie, systems that respond to the patient inspiratory effort by varying gas delivery) allow spontaneous breaths to be classified. During IMV, spontaneous breaths are pressure-controlled; pressure-, volume-, or flow-triggered; pressure-limited; and pressure-cycled (Table 1). Using the proposed system allows a more succinct description. Mandatory breaths are pressure- or volume-/flow-controlled, machine-triggered, and machine-cycled. Spontaneous breaths are pressure-controlled, patient-triggered, and patient-cycled. As in the previous examples, the term does not allow the clinician to ascertain how mandatory breaths are controlled and fails to convey all of the important information.

Synchronized Intermittent Mandatory Ventilation (SIMV)

Definition—SIMV is a version of IMV in which the ventilator creates a timing window around the scheduled delivery of the mandatory breath and attempts to deliver the breath in concert with the patient's inspiratory effort.[4-7,15,16] If no inspiratory effort occurs during this time, the ventilator delivers the mandatory breath at the scheduled time.

Other Terms—The term SIMV appears to be universally accepted, although the first description of this mode named it intermittent demand ventilation (IDV).[15]

Manufacturers' Terms—All manufacturers who offer SIMV refer to it as such.

Chatburn's Classification—In this system, the classification is identical to that of IMV, except that mandatory breaths can be machine- or patient-triggered. During SIMV, mandatory breaths are pressure- or volume-/flow-controlled; time-, pressure-, flow-, or volume-triggered; pressure-, volume-, or flow-limited; and pressure-, volume-, flow-, or time-cycled. Spontaneous breaths are pressure-controlled; pressure-, volume-, or flow-triggered; pressure-limited; and pressure-cycled (Table 1). The proposed classification describes mandatory breaths during SIMV as pressure- or volume-/flow-controlled; machine- or patient-triggered; and machine-cycled. Spontaneous breaths are classified as pressure-controlled, patient-triggered, and patient-cycled (Table 2). Because of the synchronization process, SIMV is not possible with only a continuous-flow source. Some authors describe demand-flow IMV and continuous-flow IMV as different modes, which is clearly not the case. Although the implications for the respiratory care practitioner are quite different, the fundamental operation is the same (Fig. 5).

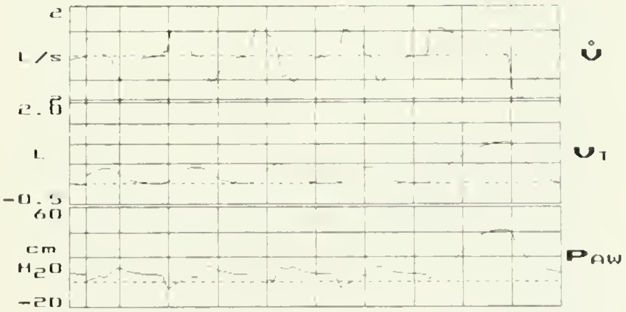

Fig. 5. Flow, volume, and pressure waveforms for what is commonly referred to as SIMV. Based on the Chatburn classification,[1] this is volume-controlled intermittent mandatory ventilation, combined trigger (ie, mandatory breaths are either machine- or patient-triggered). Conventional IMV would be considered VC-IMV, machine-triggered because no synchronization (patient-triggering of mandatory breaths) is possible.

Continuous Positive Airway Pressure (CPAP)

Definition—CPAP is a mode of ventilator operation in which a clinician-set level of pressure is maintained constant, while the patient is allowed to breathe spontaneously.[4-7,17]

Other Terms—Few processes have garnered the virtual avalanche of abbreviations and acronyms heaped upon CPAP. While differences do exist, the following have all been used to describe or have been used interchangeably with CPAP: positive end-expiratory pressure (PEEP), end-expiratory pressure (EEP), inspiratory positive airway pressure (IPAP), expiratory positive airway pressure (EPAP), continuous distending pressure (CDP), and continuous positive pressure breathing (CPPB). The most common explanation of the difference between PEEP and CPAP is that PEEP is elevated baseline pressure during mechanical ventilation, whereas CPAP is elevated baseline pressure during spontaneous breathing. This explanation falls short when IMV is used, because an elevated baseline pressure is used after both spontaneous and mandatory breaths (Fig. 6). Perhaps the best way to differentiate between the two is that CPAP is, as we are discussing, a mode of ventilator operation, whereas PEEP is simply control of baseline pressure during use of a separate mode of ventilation. On some occasions, CPAP has been described as IMV with a rate of 0.

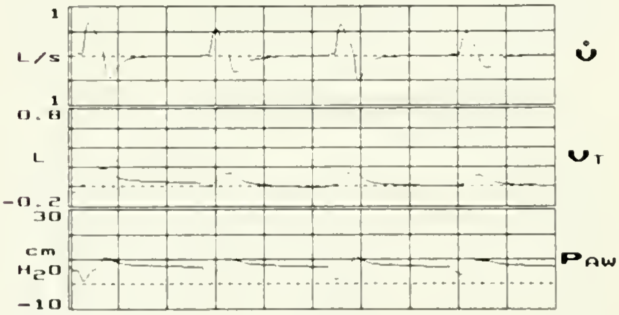

Fig 6. Flow, volume, and pressure waveforms for continuous positive airway pressure (CPAP). All breaths are spontaneous, and under the Chatburn classification system are to be called constant airway pressure (CAP), which, like pressure support, is under a larger heading of continuous spontaneous ventilation.

Manufacturers' Terms—The term CPAP is used by all manufacturers to describe this mode. In some instances, there is a control labeled CPAP, whereas in others the mode is accessed via the "spontaneous" mode. In both cases, the level of end-expiratory pressure is selected using a baseline or PEEP/CPAP control.

Chatburn's Classification—Because CPAP is devoid of mandatory breaths, only the spontaneous breaths need be considered. Spontaneous breaths are pressure-controlled; pressure-, flow-, or volume-triggered; pressure-limited; and pressure-cycled (Table 1). More simply, CPAP is pressure-controlled, patient-triggered, patient-cycled, unsupported spontaneous breathing.

Pressure-Controlled Ventilation (PCV)

Definition—The term PCV generally refers to CMV in which all breaths are pressure-limited and time-cycled.[4-7,18] This usually means CMV in the truest sense. No patient-triggering is possible. In reality, PCV refers to how the mandatory breaths are "controlled" rather than specifying a mode. This is particularly evident when you realize that pressure-controlled (pressure-limited, time-cycled mandatory breaths) could be delivered in CMV, A/C, or IMV.

Other Terms—PCV is usually referred to as such, although ventilation of neonates is accomplished with this mode every day and called simply IMV. Some authors refer to PCV as pressure-limited ventilation or more specifically as pressure-limited, time-cycled ventilation.

Manufacturers' Terms—On the whole, manufacturers label PCV as PCV. The Hamilton Veolar[8] allows either PC-IMV or PC-CMV to be selected, while the Puritan-Bennett 7200ae[2] allows PCV to be selected as a breath-delivery technique with mode selected separately. The PPG IRISA allows PCV in either the IMV or CMV mode, designating the mode as "IMV + Pressure-Limited" in the display window.

Chatburn's Classification—The term pressure-controlled ventilation, of itself, only serves to describe how mandatory breaths are delivered. PC-CMV (Fig. 7), PC-AMV, and PC-IMV (Fig. 8) provide more specific information and allow any of these to be classified. During PC-CMV, mandatory breaths are pressure-controlled, time-triggered, pressure-limited, and time-cycled. The fact that breaths are pressure-controlled precludes them from being flow-/volume-cycled or pressure-cycled (pressure is the limit variable); so, time is the only possible cycling variable. The other PC modes can be classified by selecting the previous classifications and changing the mandatory breath descriptions to pressure-controlled, pressure-limited, and time-cycled, with the trigger unchanged. These classifications are shown in Tables 1 and 2.

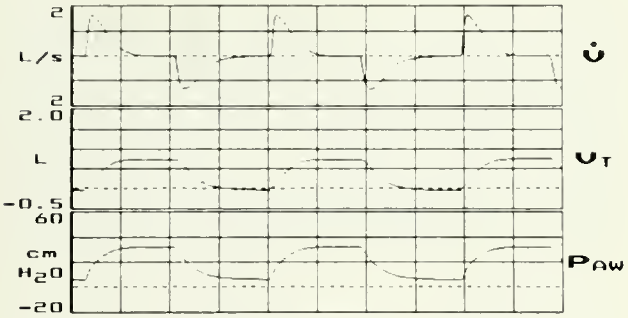

Fig. 7. Flow, volume, and pressure waveforms for pressure-controlled continuous mandatory ventilation (PC-CMV). The term pressure-controlled in its present usage tells the practitioner only that mandatory breaths are pressure preset and hence volume variable. The picture here can be further classified as PC-CMV, machine-triggered.

Pressure-Controlled Inverse-Ratio Ventilation (PCIRV)

Definition—PCIRV is a particular version of PC-CMV in which all breaths are pressure-limited and time-cycled, and the patient cannot initiate an inspiration.[4-7,19-23] Additionally, as the name implies, inspiration is longer than expiration.

Other Terms—PCIRV is sometimes simply shortened to IRV.

Manufacturers' Terms—No manufacturer has labeled a mode as PCIRV. In most instances PCIRV is initiated by selecting the PCV mode and adjusting settings to provide the desired I:E.

Chatburn's Classification—The mode termed PCIRV can be classified as pressure-controlled, time-triggered, pressure-limited, and time-cycled. All breaths are mandatory. The proposed classification refers to PCIRV as pressure-controlled, machine-triggered, pressure-limited, and machine-cycled (Fig. 9). These descriptions should lead the reader to question why PCIRV is considered a separate mode. The only difference between PCIRV and PCV is the I:E. Volume-oriented modes are not classified separately with respect to I:E. Why should pressure-oriented modes be?

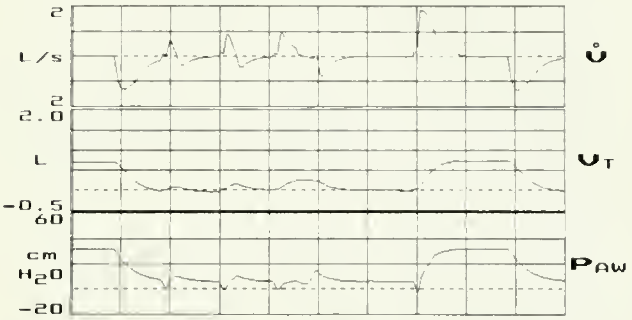

Fig. 8. Flow, volume, and pressure waveforms for pressure-controlled intermittent mandatory ventilation, combined trigger. Note the negative pressure deflection prior to mandatory breath delivery.

Airway Pressure Release Ventilation (APRV)

Definition—APRV is often described as two levels of CPAP that are applied for set periods of time and that allow spontaneous breathing to occur at both levels. This mode is said to allow the clinician to set the two CPAP levels (known as CPAP and release pressure) and the time spent at each level.[4,5,24]

Other Terms—APRV has been referred to as bilevel airway pressure (BiPAP), variable positive airway pressure (VPAP), and CPAP with release.

Manufacturers' Terms—The PPG IRISA is the only ventilator that offers APRV and uses that terminology. Respironics has a modified nasal CPAP system capable of providing APRV. Both the device and the mode are called BiPAP.

Chatburn's Classification—Scrutiny of the APRV pressure, volume, and flow waveforms reveals its similarity to PCIRV. In fact, if spontaneous breathing is absent, the two modes are indistinguishable. Mandatory breaths (which occur when the pressure rises from low pressure to higher pressure) are pressure-controlled, time-triggered, pressure-limited, and time-cycled. Spontaneous breaths are pressure-controlled, pressure-triggered, pressure-limited, and pressure-cycled during APRV with the IRISA (Fig. 10). In the original description by Downs and Stock, a continuous flow of gas was used; therefore, spontaneous breaths were not controlled (Fig. 11). This mode demonstrates the strength of this classification system. While the proponents of APRV speak of bilevel CPAP and dropping from CPAP to release pressure,[25-28] applying the classification principles unmasks the black box. APRV is no different from the mode of ventilation that has been provided by conventional neonatal ventilators for more than 20 years.

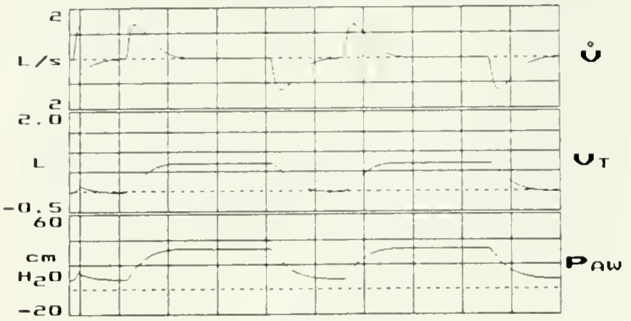

Fig. 9. Flow, volume, and pressure waveforms for pressure-controlled inverse-ratio ventilation (PCIRV). Compared to Figure 6, the only difference is that inspiratory time is greater than expiratory time. We suggest that PCIRV is not a new mode but simply pressure-controlled, continuous mandatory ventilation, machine-triggered (PC-CMV-MT).

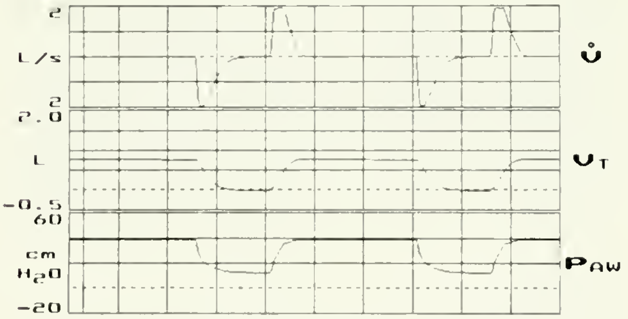

Fig. 10. Flow, volume, and pressure waveforms for airway pressure release ventilation (APRV) without patient spontaneous breathing. Note the similarity to Figure 8.

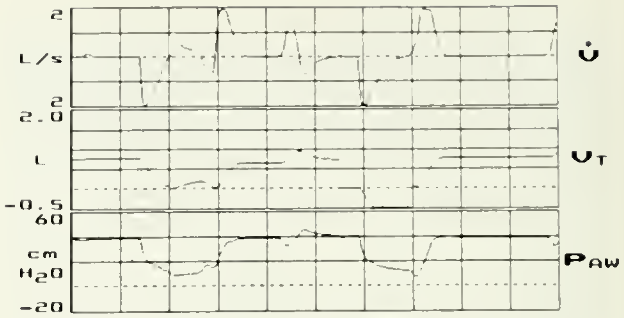

Fig. 11. Flow, volume, and pressure waveforms of APRV demonstrating spontaneous breathing throughout the ventilatory cycle. Based on the Chatburn classification, APRV is, as performed by the IRISA, pressure-controlled intermittent mandatory ventilation, combined trigger. The system described by Stock and Downs provides only machine-triggered breaths in this mode.

BiPAP—The term BiPAP is both a purported mode of ventilation and the trademark name of a device. When described as a mode, BiPAP is analogous to APRV. However, the BiPAP device is also capable of delivering pressure support ventilation.[29] This example further serves to illustrate the confusion created by manufacturers and reinforced by clinicians.

Pressure Support Ventilation (PSV)

Definition—Pressure support ventilation is a mode of ventilator operation in which the patient's inspiratory effort is assisted by the ventilator up to a preset level of inspiratory pressure. Inspiration is terminated when peak inspiratory flowrate reaches a minimum level or a percentage of initial inspiratory flow (Fig. 12). This allows the patient to

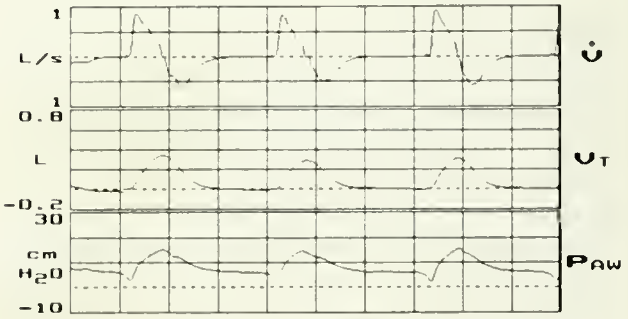

Fig. 12. Flow, volume, and pressure waveform for pressure support ventilation. Using the Chatburn classification, all breaths during PSV are spontaneous and pressure-controlled; thus PSV is pressure-controlled continuous spontaneous ventilation (PC-CSV).

determine his own rate, inspiratory time, and tidal volume.[4,5,30-32]

Other Terms—PSV suffers a fate similar to that of CPAP in the variations of its name. The literature refers to PSV as inspiratory assist (IA), inspiratory pressure support (IPS), spontaneous pressure support (SPS), and inspiratory flow assist (IFA).

Manufacturers' Terms—All manufacturers have different algorithms for the provision of pressure support, but all label it PSV. Unfortunately, the PSV mode is often invoked through the spontaneous mode control, leading some to believe that the ventilator is not providing positive pressure ventilation.

Chatburn's Classification—According to the definitions of spontaneous and mandatory breaths, all PSV breaths are spontaneous. Therefore, PSV can be classified as pressure-controlled; pressure-, flow-, or volume-triggered; pressure-limited; and flow-cycled. The proposed classification is pressure-controlled, patient-triggered, pressure-limited, patient-cycled ventilation.

Mandatory Minute Ventilation (MMV)

Definition—MMV is a mode of ventilator operation that allows the patient to breathe spontaneously, yet ensures that a minimum level of minute ventilation ($\dot{V}_E$), set by the clinician, is always achieved.[4,5,33-35] This can be accomplished by the use of increasing levels of PSV (Hamilton Veolar)[8] or by delivery of mandatory breaths (Bear 5,[3] IR-ISA,[36] Ohmeda Advent[37]).

Other Terms—MMV has been called minimum minute volume (MMV), augmented minute volume (AMV), and extended mandatory minute ventilation (EMMV).

Manufacturers' Terms—The initial description of MMV was termed mandatory minute volume, but ventilator manufacturers use all the terms listed above (EMMV, MMV, AMV).

Chatburn's Classification—MMV is one of the modes in which the conditional variable (in this case $\dot{V}_E$) is critically important to classification. If spontaneous breathing is used, then breaths are pressure-controlled; pressure-, flow-, or volume-triggered; pressure-limited; and pressure-cycled. As long as the conditional variable is met, this system does not change. If $\dot{V}_E$ falls below the preset minimum, classification depends upon the ventilator used. With the Veolar, breaths are assisted with increasing levels of PSV. In this instance, there are still no mandatory breaths. Therefore, MMV is pressure-controlled; pressure-, flow-, or volume-triggered; pressure-limited; and flow-cycled (Fig. 13). The proposed classification is pressure-controlled, patient-triggered, pressure-limited, patient-cycled ventilation. With the other ventilators, if the conditional variable is not met, mandatory breaths are delivered. This creates an IMV-like situation in which both spontaneous and mandatory breaths must be described (Fig. 14). Spontaneous breaths are classified identically to CPAP or PSV, depending upon how the clinician adjusts ventilator settings. Mandatory breaths are volume-/flow-controlled, time-triggered, flow- or volume-limited, and time- or flow-cycled. More simply, mandatory breaths are volume-/flow-controlled, machine-triggered, and machine-cycled.

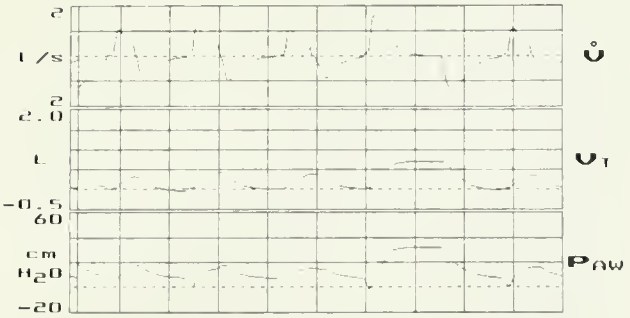

Fig. 13. Flow, volume, and pressure waveforms for mandatory minute ventilation with pressure control of spontaneous breaths. When the conditional variable ($\dot{V}_E$) is not met, the level of pressure support delivered changes. No mandatory breaths are delivered. This may also be thought of as MMV + PSV.

Combining Modes

Acknowledging Combinations

Modes of ventilator operation do not have to be used in isolation. Although certain modes have to

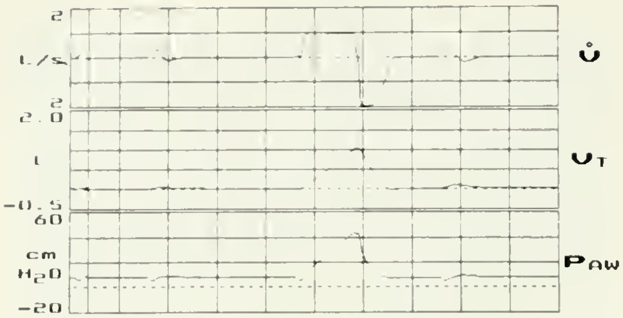

Fig. 14. Flow, volume, and pressure waveforms for mandatory minute ventilation with volume control of mandatory breaths. When the conditional variable is not met, a volume-controlled, mandatory breath is delivered. This may also be thought of as MMV + IMV.

stand alone based upon their function, others can be combined. We have previously discussed the combination of IMV with CPAP and PCV with IMV. Essentially, any mode that provides for both spontaneous breathing and mandatory breaths can be combined with a mode that uses spontaneous breathing. For instance, PSV can be combined with IMV but not with CMV. In these cases, creation of a new term to describe the combined modes is undesirable. It is simpler and more descriptive to acknowledge the contributions of each mode (ie, IMV + PSV, IMV + CPAP, PC-IMV, or VC-IMV + PSV, Fig. 15).

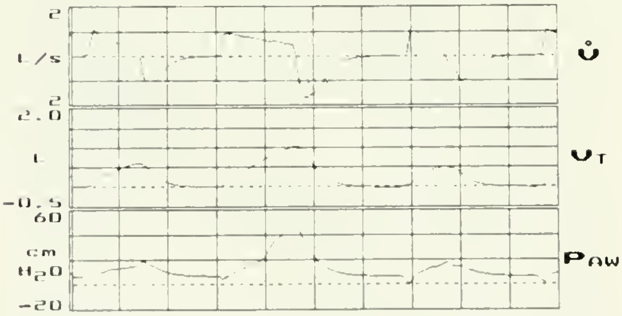

Fig. 15. Flow, volume, and pressure waveforms for volume-controlled intermittent mandatory ventilation plus pressure support ventilation. Using two modes together does not constitute a new mode.

Volume-Assured Pressure Support (VAPS)

Definition—VAPS is one of the many new modes of ventilation dependent upon conditional variables and ventilator logic. This mode was introduced by Bird Products on the 8400ST and allows the patient to be placed in the PSV mode with a minimum V_T/breath assured.[38] If during a PSV breath, the ventilator ascertains that the patient is not receiving the minimum V_T, flowrate is held constant and pressure is allowed to increase until the desired volume is reached. These modes use the "if, then" type of logic. In many ways VAPS is like MMV, only on a breath-to-breath rather than on a minute-to-minute basis.

Other Terms—Most applications of VAPS are recently developed manufacturer-supplied enhancements not identified by other names.

Manufacturers' Terms—Siemens has introduced two new 'modes' known as pressure-regulated volume-control and volume-support.[39] Both modes adjust parameters based on a conditional variable.

Chatburn's Classification—The VAPS mode simply provides varying levels of PSV controlled by a conditional variable (V_T). If the conditional variable is met, the classification is identical to that of PSV. If the conditional variable is not met, then the VAPS breaths are pressure-controlled (conditional variable met) or volume-/flow-controlled (conditional variable not met); pressure-, flow-, or volume-triggered; pressure-limited and flow- (conditional variable met) or volume-cycled (conditional variable not met). Because these adjustments are made on a breath-to-breath basis, both kinds of breaths may be seen in the same minute. We prefer to refer to this mode as adaptive pressure support.

Taking Another Look

Over the past quarter century of mechanical ventilation, modes have been introduced without scientific testing and named by inventors and marketing executives without any consistency or regard for users. If the classification system discussed here is applied, it is clear that many new modes are, in fact, not new, and that most modes can fit into one of a few basic categories (Fig. 16). We suggest the following steps for defining and naming modes and hope to convince the reader that improvements are needed.

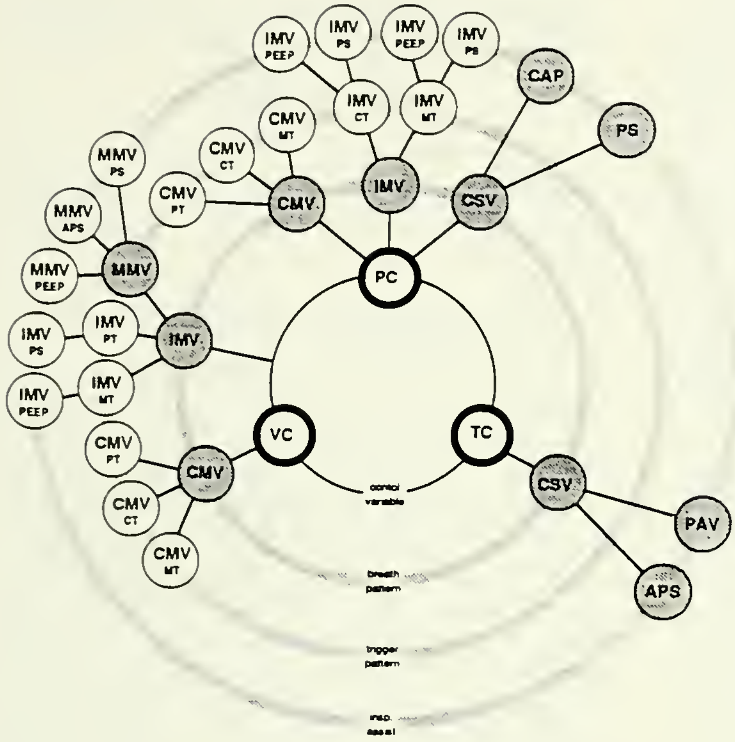

Fig. 16. The constellation of ventilator modes. The first step in describing a mode is to determine the control variable (dark rings). Volume and flow control have been made one for simplicity. Using the combination of phase and conditional variables and breath pattern, the root modes are described (shaded circles). The third generation of circles describes the trigger pattern for mandatory breaths and allows description of the root-mode variations. The fourth generation separates versions by inspiratory support of the spontaneous breaths. (ie, IMV + PEEP is unsupported spontaneous breathing and IMV + PS is supported spontaneous breathing). This fourth generation helps describe mode combinations. For clarity, volume-controlled (preset) IMV is on the line between VC and PC because mandatory breaths are volume-controlled, whereas spontaneous breaths are pressure-controlled. This system is meant to complement the classification system and attempts to show the relationship between the modes and their clinically important distinctions.

Step 1: Determining the Control Variable—Volume- vs Pressure-Controlled

Identification of the control or preset variable for mandatory breaths is the first distinction to be made regarding mode. If only spontaneous breaths are used, then the control variable for those breaths should be determined. This preset variable serves as a prefix to further mode description, using phase and conditional variables. In modes in which mandatory breaths are employed, the control variable distinction describes breath delivery and can be thought of as a modifier of the mode. Chatburn's classification system allows volume, pressure, flow, and time control; yet, for practical purposes volume and flow control can be placed together. Therefore the first step in classifying an operational mode is determining whether it is volume-/flow-, pressure-, or time-controlled.

Looking back at Tables 1 and 2, it is easy to see the complexity surrounding operational mode terminology. We propose that initially distinguishing the control variable for the mode immediately decreases the number of modes by five. For instance, when a clinician says the patient is on CMV, you may or may not assume that this means volume-controlled CMV. Of course it might mean pressure-controlled CMV, but except for the control variable distinction the mode is still CMV. This allows PCV, PC-IMV, PC-SIMV, PC-IRV, and APRV to be removed from the chart. Although the prefix tells the clinician what variable is controlled, the mode (as described by phase and conditional variables) remains unchanged. So, when it is reported that the patient is on CMV, you can ask, volume-controlled or pressure-controlled? Both modes are the same; it is the control variable that distinguishes between the two types of CMV.

Step 2: Determining Phase Variables

The modes are more precisely described by determining the specific combination of phase variables that constitutes each one. By defining the current modes previously, we can come to agreement as to which abbreviations are useful to add to the vernacular to describe the mode of operation. The term IMV is quite descriptive (intermittently mandatory breaths are delivered) and is a good starting point for determining future names. If IMV is intermittent, then CMV should be "continuous mandatory" not "controlled mechanical" ventilation. This type of logic preserves the familiar abbreviation while providing a more consistent and descriptive name.

In Table 1, it can be seen that the modes PCV and PCIRV use exactly the same control and phase variables. Yet, they are often described as two different modes simply because with PCIRV inspiratory time is longer than expiratory time. But, when the I-E ratio is reversed in the volume-control mode, a separate distinction is not made. We provide this as further support for abandoning the term PCIRV as a mode.

A similar comparison can be made for IMV and SIMV. The only difference between these two modes of ventilation is that during SIMV, the trigger variable for mandatory breaths can be either machine- (time) triggered or patient- (pressure-, volume-, flow-) triggered. We suggest that this difference does not constitute a change in mode but simply a change in one of the phase variables.

To carry this further, the only difference between CMV, A/C, and AMV is the method of triggering. In CMV all breaths are machine-triggered; in A/C breaths can be either machine- or patient-triggered; and in AMV all breaths are patient-triggered. Based on this single difference, we suggest that these three represent one mode. However, the distinction of patient-triggering is important, so we suggest that these modes be called CMV-MT (continuous mandatory ventilation-machine triggered), CMV-CT (continuous mandatory ventilation-combined trigger), and CMV-PT (continuous mandatory ventilation-patient triggered). We understand that changing terminology may present a burden for those accustomed to the old way, but we feel the new terminology will be easier to learn because of its logic.

Table 3 represents what we believe are the basic modes of ventilator operation, their classification based upon control and phase variables, and a list comparing the old terminology with the proposed terminology. Table 4 demonstrates how a specific grouping of control phase variables can describe variations of the modes. Figure 3 demonstrates the branching of modes from the three control variables to the primary modes and their offspring.

New Terminology

In an effort to preserve as much useful terminology as possible, we suggest that all modes of ventilator operation be described by the following terms.

Continuous Mandatory Ventilation (CMV)

CMV has been called controlled mechanical ventilation, assist/control, and assisted mechanical ventilation. Preservation of the abbreviation maintains the familiar CMV while the terms continuous and mandatory are more descriptive of the process. All breaths in the CMV mode are mandatory by definition and are continuously available (as opposed to intermittently available as in IMV). CMV, then, is one of the root modes that can be modified by the control variable and the phase variables. If breaths are pressure preset, then the mode is termed pressure-controlled, continuous mandatory ventilation (PC-CMV). For volume preset ventilation, the term volume-controlled, continuous mandatory ventilation (VC-CMV) is used. The other variations of CMV relate to triggering. Assist/control has been used to designate the fact that the patient can trigger mandatory breaths between machine-triggered breaths. Rather than use this unrelated term (A/C), we suggest that the distinction be identified (ie, machine-triggered, MT; patient-triggered, PT; and combined trigger, CT). Therefore, a mode of ventilator operation that is pressure preset, that delivers only mandatory breaths (patient- or machine-triggered) is termed pressure-controlled, continuous mandatory ventilation, combined-trigger (PC-CMV-CT). The six possible combinations of control and trigger variables derived from the root mode CMV are shown in Table 4.

Intermittent Mandatory Ventilation (IMV)

The term IMV is the old term, but it adequately describes the mode. In IMV, mandatory breaths are delivered intermittently, allowing the patient to breathe spontaneously between them. The derivatives of the root-mode IMV, then, are based upon control and mandatory breath-trigger variables. If volume is preset and all mandatory breaths are ma-

Table 3. Suggested Terminology for Operational Modes, Their Classification, and Relationship to Old Terminology

	Mandatory				Spontaneous					Control Logic		
Mode	Control	Trigger	Limit	Cycle	Control	Trigger	Limit	Cycle	Supported?	Conditional Variable	Action	Prior Terms
Constant airway pressure (CAP)	—	—	—	—	Pressure	Pressure, volume, or flow	Pressure	Pressure	No	—	—	CPAP
Continuous spontaneous ventilation (CSV)	—	—	—	—	Pressure	Pressure, volume, or flow	Pressure	Volume	Yes	—	—	PSV
Continuous mandatory ventilation (CMV)	Pressure	Pressure, volume, flow, or time	Pressure	Time	—	—	—	—	—	Time or patient effort	Machine-to-patient trigger	PC-CMV, PCIRV, PC-A/C
	Volume/ flow	Pressure, volume, flow, or time	Volume/ flow	Volume, flow, or time	—	—	—	—	—	Time or patient effort	Machine-to-patient trigger	CMV, A/C
Intermittent mandatory ventilation (IMV)	Pressure	Pressure, volume, flow, or time	Pressure	Time	Pressure	Pressure, volume, or flow	Pressure	Pressure	No	Time or patient effort	Machine-to-patient trigger	PC-IMV, APRV, BiPAP, PC-SIMV
	Volume/ flow		Volume or flow	Volume, flow, or time	Pressure	Pressure, volume, or flow	Pressure	Pressure	No	Time or patient effort	Machine-to-patient trigger	IMV, SIMV
Mandatory minute ventilation (MMV)	Volume/ flow	Time	Volume or flow	Volume, flow, or time	Pressure	Pressure, volume, or flow	Pressure	Pressure	Yes*	Minute volume or time	Spontaneous-to-mandatory breath	MMV, EMMV
Proportional assist ventilation (PAV)	—	—	—	—	Time	Volume or flow	Volume or flow	Volume or flow	Yes	—	—	—

*Optional.

Table 4. The Basic Modes of Operation and Their Derivatives

	Mandatory Breath				Spontaneous Breath					
Mode	Control	Trigger	Limit	Cycle	Control	Trigger	Limit	Cycle	Supported?	Proposed Terminology
Continuous mandatory ventilation (CMV)	Pressure	Machine	Pressure	Machine	—	—	—	—	—	Pressure-controlled CMV, machine-triggered
	Volume or flow	Machine	Volume	Machine	—	—	—	—	—	Volume-controlled CMV, machine-triggered
	Pressure	Patient	Pressure	Machine	—	—	—	—	—	Pressure-controlled CMV, patient-triggered
	Volume or flow	Patient	Volume	Machine	—	—	—	—	—	Volume-controlled CMV, patient-triggered
	Pressure	Patient or machine	Pressure	Machine	—	—	—	—	—	Pressure-controlled CMV, combined trigger
	Volume or flow	Patient or machine	Volume	Machine	—	—	—	—	—	Volume-controlled CMV, combined trigger
Intermittent mandatory ventilation (IMV)	Pressure	Machine	Pressure	Machine	Pressure	Patient	Pressure	Patient	No	Pressure-controlled IMV, machine-triggered
	Volume or flow	Machine	Volume	Machine	Pressure	Patient	Pressure	Patient	No	Volume-controlled IMV, machine-triggered
	Pressure	Machine or Patient	Pressure	Machine	Pressure	Patient	Pressure	Patient	No	Pressure-controlled IMV, combined trigger
	Volume or flow	Machine or patient	Volume	Machine	Pressure	Patient	Pressure	Patient	No	Volume-controlled IMV, combined trigger
Continuous spontaneous ventilation (CSV)	—	—	—	—	Pressure	Patient	Pressure	Patient	Yes	CSV or pressure support ventilation
Mandatory minute ventilation (MMV)	Volume or flow	Machine	Volume	Machine	Pressure	Patient	Pressure	Patient	No	MMV, volume-controlled
	—	—	—	—	Pressure	Patient	Pressure	Patient	Yes/no	MMV, pressure-controlled
Proportional assist ventilation (PAV)	—	—	—	—	Time	Patient	Volume or flow	Patient	Yes	PAV
Constant airway pressure (CAP)	—	—	—	—	Pressure	Patient	Pressure	Patient	No	CAP

chine-triggered, then the term volume-cycled, intermittent mandatory ventilation, machine-triggered (VC-IMV-MT) is used. If pressure is controlled and machine- or patient-triggering of mandatory breaths is possible (formerly SIMV), then this mode is called pressure-controlled, intermittent mandatory ventilation, combined-trigger (PC-IMV-CT). Table 4 demonstrates the other two derivatives of the root-mode IMV.

Continuous Spontaneous Ventilation (CSV)

Formerly termed pressure support ventilation, this mode sustains the greatest change suggested. However, the terms are congruent with the previous two modes. The mode we know as PSV is composed solely of spontaneous breaths (by our definition earlier in the paper), and each breath is supported to the same extent, similar to CMV. Hence, the logical terminology is continuous spontaneous ventilation. CSV can be combined with other modes to form a combined mode such as VC-IMV-PT + CSV (ie, volume-controlled, intermittent mandatory ventilation, patient-triggered plus continuous spontaneous ventilation). There are three types of CSV. The first we continue to call pressure support ventilation and define as previously discussed. The old term CPAP is a variation of CSV, differing from PSV only in that during PSV breaths are supported, and in CPAP, they are unsupported. The third type of CSV we refer to as adaptive pressure support (APS). Essentially, CSV-APS is pressure support ventilation with a conditional variable (usually tidal volume). If, during the course of continuous spontaneous ventilation, the volume delivered by the ventilator is less than the preselected tidal volume, the ventilator maintains the current flowrate until the desired volume is achieved. This means that the ventilator changes from pressure-controlled to volume-controlled at mid-inspiration. Therefore, all breaths remain spontaneous; only the control variable changes. It may be helpful to think of APS as a breath-to-breath version of MMV.

Mandatory Minute Ventilation (MMV)

This mode also retains its original abbreviation and one of its meanings. A preset minute ventilation (conditional variable) is guaranteed by the ventilator. Most commonly this is accomplished by delivery of a machine-triggered, volume-preset mandatory breath at appropriate intervals. However, the Hamilton Veolar assures the minute ventilation by increasing the CSV (PSV) pressure limit. In Table 4, we note these differences and suggest that MMV might have two derivatives, PC-MMV and VC-MMV—or MMV + PSV and MMV + IMV.

Proportional Assist Ventilation (PAV)

PAV has been described by Younes et al,[40-42] but has yet to be incorporated into any commercially available equipment. We mention it here for the sake of completeness. PAV delivers gas based upon simultaneous changes in airway flow and volume and a gain setting on the machine. The greater the patient's demand, the greater support the ventilator provides. It is our contention that PAV, while similar to CSV (PSV) in theory, is quite different with respect to control and phase variables.

Constant Airway Pressure (CAP)

Constant airway pressure (CAP) was formerly continuous positive airway pressure (CPAP). Most of the abbreviation is spared in this terminology change, but the use of the term constant more aptly describes the airway pressure waveform. Additionally, the term continuous has heretofore been associated with mandatory breaths or machine support of spontaneous breaths. CAP, then, refers to pressure-controlled, patient-triggered, pressure-limited, patient-cycled, unsupported spontaneous breathing.

In Summary

These changes are proposed as a starting point for the more logical application of ventilator terminology and are for your consideration and debate. It is our contention that this system can be easily adopted once the basics of the classification system are understood.

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Essential Gas Delivery Features of Mechanical Ventilators

Robert M Kacmarek PhD RRT

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- II. **Modes of Ventilation**
- III. **A/C versus SIMV**
- IV. **Pressure-Targeted Ventilation**
- V. **MMV, APRV, BiPAP, and PAV**
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- IX. **PEEP/CPAP and F_{IO_2}**
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Introduction

The sophistication and capabilities of mechanical ventilators have greatly expanded over the last 10 years. As a result of the incorporation of microprocessors into many of the newest generation of ventilators, virtually any conceivable gas-flow delivery pattern is possible. Users of this technology must ask, Is all of the added versatility clinically efficacious, and is the cost-benefit relationship favorable enough to justify the expense of these newer systems?

In this paper, I review the specific gas delivery features incorporated into today's mechanical ventilators and classify those features as essential, recommended, and optional, and identify necessary ranges for selected variables. Mechanical ventilators for intensive care, transport, and home care are addressed.

Modes of Ventilation

Of all specific features of mechanical ventilators, the one that probably engenders the most controversy is mode of ventilation. Unfortunately, in spite of volumes of studies attempting to evaluate and define appropriate modes, no outcome data are available to guide practitioners in their choice of mode. As a result, the efficiency and efficacy of any particular mode has been established only on the basis of discussions of the technologic capability of the mode and the real and theoretical alterations in physiologic response associated with the use of the particular mode. In most cases, arguments regarding the benefits derived from a particular mode are based on anecdotal reports and the individual management philosophies of clinicians.

Dr Kacmarek is Assistant Professor, Department of Anesthesiology, Harvard Medical School, and Director, Respiratory Care Services, Massachusetts General Hospital—Boston, Massachusetts.

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Reprints: Robert M Kacmarek PhD RRT, Respiratory Care, Ellison-4, Massachusetts General Hospital, Boston MA 02114.

In general, most would agree that approaches to ventilatory support can be categorized into two groups—full ventilatory support (FVS) and partial ventilatory support (PVS),[1,2] with FVS being defined as an approach to ventilatory support in which the ventilator performs the majority, if not all, of the work of breathing. That is, the ventilator controls the rate, depth, and pattern of gas delivery. On the other end of the spectrum, PVS defines an approach to ventilation in which the patient and the machine are allowed to more intimately interact to determine the gas delivery methodology (that is, the patient and the machine share the work of breathing). At least theoretically, it seems reasonable that ventilators, particularly those in the ICU, should be able to provide both FVS and PVS, with the most ideal mode being that with the potential of allowing movement across the complete spectrum from spontaneous breathing to PVS to FVS.

Table 1 lists an abbreviated comparison of modes of ventilation, based on their ability to provide volume- or pressure-targeted ventilation and to provide PVS and FVS. Note that pressure-targeted approaches equivalent to all classic modes are now available. Most modes, with the exception of classic control or assist/control (volume- or pressure-targeted) are capable of providing both PVS and FVS.

Table 1. Classification of Modes of Ventilation

Volume-Targeted	Pressure-Targeted	Level of Support
Volume control	Volume control	Full
Volume A/C*	Volume A/C, BiPAP	Full
Volume assist	Pressure support	Partial or full
SIMV	Pressure control,† APRV, BiPAP	Partial or full
MMV	MMV	Partial or full

* A/C = assist/control. BiPAP = bilevel positive airway pressure, depending upon setup can provide either A/C or SIMV (actually intermittent mandatory ventilation, IMV). SIMV = synchronized intermittent mandatory ventilation. APRV = airway pressure release ventilation, actually is equivalent to intermittent mandatory ventilation because a constant flow of gas is generally used. MMV = mandatory minute ventilation, depending upon specific ventilators, may be pressure- or volume-targeted.

† With some ventilators, spontaneous ventilation or pressure support ventilation is allowed between pressure-controlled breaths.

A/C versus SIMV

The controversy over which is the more appropriate mode of ventilation, assist/control (A/C) or synchronized intermittent mandatory ventilation (SIMV), has been ongoing for nearly 20 years and has never been, and probably never will be, settled by outcome studies. The studies that have been performed[3-6] have all compared the two approaches under dissimilar circumstances (ie, SIMV rate of 4/min, as compared to normal A/C rates). Differences regarding mean intrathoracic pressure changes, mean airway pressures, and levels of ventilation have been noted. However, no data to support differences in length of mechanical ventilation, ease of ventilator discontinuation, or frequency of ventilator-induced complications have been documented. As a result, it is impossible to argue cogently, based on the available scientific data, for the inclusion or exclusion of either volume-targeted A/C or SIMV on the basic ICU ventilator. (The advantages and disadvantages of each are outlined in Table 2.[1,7,8]) SIMV allows a broader range of available ventilatory support (PVS to FVS); however, A/C is firmly established in the minds of many clinicians as the ideal approach to ventilatory support. As a result, it seems reasonable to view both volume-targeted A/C and SIMV as essential for the basic ICU ventilator.

Pressure-Targeted Ventilation

Over the last 5 years, much of the discussion regarding modes of mechanical ventilation has centered on the use of pressure-targeted approaches. Of all the modes of ventilation currently available, pressure support ventilation (PSV) is the approach that has probably received the most attention in clinical studies.[9-12] As a result of the work of breathing (WOB) imposed by endotracheal tubes,[13,14] humidification systems,[15] ventilator demand valves,[15] and PEEP/CPAP devices,[15] low levels of pressure support (5-15 cm H_2O) have been recommended for use on most, if not all, patients being ventilated in SIMV or in CPAP.[16] As demonstrated by Brochard et al[10] (Table 3), by ad-

Table 2. Volume-Targeted Assist/Control (A/C) vs Synchronized Intermittent Mandatory Ventilation (SIMV)

Mode	Description	Advantages	Disadvantages
A/C	Volume control; however, spontaneous breathing can increase rate; V_T* constant; peak pressure variable; FVS only.	All breaths at targeted V_T; patient may exceed set rate; control over minimum MV; volume control possible with sedation, sedation/paralysis, or hyperventilation.[1]	Rapid breathing may cause respiratory alkalosis, air-trapping auto-PEEP, cardiovascular compromise; peak airway pressure variable.
SIMV	Volume A/C with spontaneous breathing via demand system between volume breaths; PVS to FVS.	Able to provide any level of ventilatory support; allows near complete spontaneous ventilation; at low rates, air-trapping, auto-PEEP, respiratory alkalosis, and cardiovascular compromise less likely than with A/C;[7] can provide FVS with sedation/paralysis, or hyperventilation.	Stacking of spontaneous breaths on mechanical breaths; dyssynchrony as a result of differing gas delivery patterns; increased WOB at low mandatory rates,[8] may cause air-trapping, auto-PEEP, and respiratory alkalosis at rapid spontaneous rates; peak airway pressure variable.

*V_T = tidal volume; FVS = full ventilatory support; MV = minute volume; PVS = partial ventilatory support; and WOB = work of breathing.

justing the PSV level, it is possible to titrate the amount of work performed by the spontaneously breathing patient to a greater extent than is possible by any other commonly available mode. Although no one has been able to demonstrate a difference in outcome during various approaches to weaning,[17-19] Hurst et al[12] have shown that the addition of low levels of pressure support (a mean level of 9 cm H_2O) to SIMV dramatically improves gas exchange, dead-space-to-tidal-volume ratio, and ventilatory pattern during weaning (Table 4). If properly applied to spontaneously breathing patients, PSV improves patient comfort, decreases imposed WOB, eliminates accessory muscle use, and establishes a more normal ventilatory pattern.[9-12,16] As a result of these data and the virtual universal use of pressure support, it must be considered essential on the basic ICU ventilator.

More recently, controversy over the benefits of pressure-control ventilation (PCV) vs volume-control ventilation (VCV) has surfaced in the medical literature.[20,21] (A comparison of PCV vs VCV is provided in Table 5.[22]) A number of reports of uncontrolled studies have indicated that PCV, particularly when the I-E ratio is inverted, is of significant benefit when applied during adult respiratory distress syndrome.[23-26] Unfortunately, only limited data under controlled clinical or laboratory settings support these initial reports. A single study by Abraham and Yoshihara[27] using normal I:E (1:2) indicated a statisticallly significant increase in P_{aO_2} (80-92 torr) when volume-control was changed to pressure-control. However, a number of authors in controlled patient[28-30] and animal[31] studies have been unable to demonstrate either a clinically or statistically significant improvement in gas exchange when pressure-control is used with either normal or extended I:E. As a result, the need to include this mode as one of the basic, essential modes of ICU ventilation must be questioned. In fact, the only advantage PCV has over VCV may be the fact that it maintains a constant peak airway pressure. However, if high-pressure limits are set tightly (5-10 cm H_2O above peak airway pressure), similar safety in airway pressure change can be achieved with volume-control, while constant tidal volume (V_T) delivery is maintained.[32,33] In fact, as discussed by Marini's group,[32,33] the essential feature of management of ARDS patients is the application of specific criteria limiting pressure,

Table 3. Changes in Ventilatory Variables, Gas Exchange, and Transdiaphragmatic Pressure with Increasing Levels of Pressure Support*

	Pressure Support Levels (cm H_2O)			
	0	10	15	20
V_T,† mL	377 (80)	459 (63)‡	491 (82)§	533 (131)#
RR, min^{-1}	31 (8)	29 (8)	26 (8)#	24 (7)#
$\dot{V}_E$, L · min^{-1}	11.2 (2.2)	13.0 (2.9)¶	13.1 (2.9)¶	12.3 (2.3)
P_{di}, cm H_2O	21 (12)	14 (11)#	10 (8)#	9 (8)#
$PEEP_i$, cm H_2O**	2.7 (1.7)	1.5 (1.2)¶	0.9 (1.2)¶	0.7 (0.7)¶
P_{aO_2}, torr	76 (18)	79 (19)	79 (16)	83 (19)
P_{aCO_2}, torr	45 (8)	40 (6)¶	39 (5)§	36 (6)#
$\dot{V}_{O_2}$, mL · min^{-1}	288 (49)	241 (55)§	235 (48)§	213 (54)#
$\dot{W}$, J · min^{-1}	19.5 (19.7)	13.8 (13.6)	10.9 (10.9)¶	5.0 (2.6)§
W/V, J · L^{-1}	1.58 (1.15)	0.95 (0.76)¶	0.74 (0.63)§	0.39 (0.14)#

*All data are expressed as mean (SD). Statistical analysis compares the value with pressure support zero value. (Adapted, with permission, from Reference 10.)

†V_T = tidal volume; RR = respiratory rate; $\dot{V}_E$ = minute ventilation; P_{di} = the difference between peak inspiratory and peak expiratory transdiaphragmatic pressure; $PEEP_i$ = intrinsic positive end-expiratory pressure; $\dot{V}_{O_2}$ = oxygen consumption; $\dot{W}$ = work of breathing expressed per unit of time; W/V = work of breathing per liter of ventilation.

‡$p = 0.06$.

§$p < 0.01$.

#$p < 0.001$.

¶$p < 0.05$.

**The mean value for the six patients in whom it was present.

which can be achieved with both volume- and pressure-control.

MMV, APRV, BiPAP, and PAV

Although mandatory minute ventilation (MMV), airway pressure release ventilation (APRV), bilevel positive airway pressure (BiPAP), and proportional assist ventilation (PAV) have demonstrated clinical efficacy in limited settings, they have not been demonstrated to be superior, nor can specific advantages be outlined for the use of these modes in the intubated ICU patient. It is my observation that MMV, although an attractive enhancement to SIMV, has not been embraced by the respiratory care community. The major reason for this may be a result of inadequately developed algorithms. However, in an animal model, East et al[34] have shown the benefit of adding MMV to PSV to prevent hypoventilation associated with depression of ventilatory drive.

APRV has been studied in animals,[35-37] on postoperative cardiac surgical patients,[38,39] and in patients with ARDS.[40] However, in none of these settings has it been demonstrated to be superior to conventional volume-limited A/C or SIMV.

Although BiPAP has been demonstrated to be useful in noninvasive applications,[41,42] it has not been studied in patients who require endotracheal intubation, and because it functions essentially as PSV with continuous positive (CPAP) or positive end-expiratory pressure (PEEP), it is questionable whether it can be demonstrated to be superior to their combination.

The newest and most exciting addition to the list of modes of ventilation is PAV.[43,44] This approach differs conceptually from every other available mode because it is designed to provide a greater level of ventilatory assistance when the patient demands more and a lower level of assistance when the patient demands less. Although, at least conceptually, PAV must be considered the ideal partial

Table 4. Effects of Synchronized Intermittent Mandatory Ventilation with Intermittent Pressure Support in Surgical Patients*

Value	IMV	IMV + PSV
P_{aO_2}, torr	87 (10)	99 (16)
P_{aCO_2}, torr	50 (4)	43 (5)†
RR, spontaneous, breaths/min	36 (5)	16 (3)‡
$\dot{V}_E$, L/min	13.5 ± 2	8.9 ± 1.5†
$\dot{V}_{O_2}$, mL/min	299(59)	278(68)
$\dot{V}_{CO_2}$, mL/min	270(23)	245(27)
REE, kJ/d	8,316 (714)	7,843(743)
$\bar{P}_{aw}$, cm H_2O	12 (4)	16 (5)
V_D/V_T, %	0.68 (0.1)	0.42 (0.05)†
MAP, torr	84 (7)	82 (9)
SP, torr	139(15)	130(16)

*All data are expressed as mean (SD). IMV = intermittent mandatory ventilation; PSV = pressure support ventilation; P_{aO_2} = arterial partial pressure of oxygen; P_{aCO_2} = arterial partial pressure of carbon dioxide; RR = respiratory rate; $\dot{V}_E$ = expired minute volume; $\dot{V}_{O_2}$ = oxygen consumption; $\dot{V}_{CO_2}$ = carbon dioxide production; REE = energy expenditure; $\bar{P}_{aw}$ = mean airway pressure; V_D/V_T = dead-space-to-tidal-volume ratio; MAP = mean arterial pressure; and SP = sequential pulse. (Adapted, with permission, from Reference 12.)
†$p < 0.05$.
‡$p < 0.01$.

Table 5. Volume-Targeted vs Pressure-Targeted Ventilation*

	Volume Control	Pressure Control
Rate	Set or variable	Set or variable
V_T†	Set	Variable
Peak pressure	Variable	Set
Peak flow	Set	Variable
Flow wave	Set, but variable	Set
Assist/control	Yes	Yes
Control	Yes	Yes
Inspiratory time	Variable	Variable
I-E ratio	Variable	Variable

*Reproduced, with permission, from Reference 22.
†V_T = tidal volume; I-E = inspiratory time/expiratory time.

ventilatory support (PVS) mode, its present status is purely experimental. As a result, it does not seem reasonable to consider PAV, MMV, APRV, or BiPAP as essential on the basic ICU ventilator.

Essential Modes of ICU Ventilators

Table 6 summarizes mode requirements of ICU ventilators. As noted, volume-targeted SIMV and A/C are considered essential features of the basic ICU ventilator, as is PSV. I do not believe that PCV (although desirable on a basic unit) can be considered essential. As you will note from Table 7, 3,079 patients were mechanically ventilated at the Massachusetts General Hospital (MGH) in 1991; of these, 70% were ventilated for ≤ 3 days, and 80% for ≤ 7 days. In those ventilated short-term (≤ 3 days), SIMV and PSV were the modes used almost exclusively, although volume-targeted A/C could have easily been substituted for SIMV. In patients ventilated longer than 3 days, other modes were tried—particularly pressure-control. However, only in a small percentage of this total group of patients (in the range of 2-3%) were there issues surrounding control of airway pressure. More than 95% of all the patients ventilated at MGH in 1991 were maintained with the essential modes outlined in Table 6. Although it is academically and clinically challenging to experiment with new approaches to ventilatory support, it is difficult, if not impossible, to argue that the essential features of an ICU ventilator should include those approaches to ventilation that have not been demonstrated to enhance efficacy over older modes. Further, it is fiscally irresponsible to expect that all ventilators in every ICU include every available mode of ventilation.

Table 6. Suggestions for Modes on ICU Ventilators

	Essential	Recommended	Optional
Volume Assist/Control	Yes	—	—
Volume SIMV*	Yes	—	—
Pressure Support	Yes	—	—
Pressure Assist/Control	No	Yes	—
Pressure SIMV	No	Yes	—
Pressure or Volume MMV	No	No	Yes
APRV	No	No	Yes
BiPAP	No	No	Yes

*SIMV = synchronized intermittent mandatory ventilation; MMV = mandatory minute ventilation; APRV = airway pressure release ventilation; and BiPAP = bilevel positive airway pressure.

Table 7. Patients Mechanically Ventilated at Massachusetts General Hospital in 1991

Length (days)	Number of Patients	Percentage of Total	Cumulative Percentage
≤ 1	1,523	49.5	49.5
2	379	12.2	61.7
3	202	6.6	68.3
4-7	346	11.1	79.4
8-14	255	8.3	87.7
15-21	109	3.5	91.2
22-28	83	2.7	93.9
29-56	125	4.1	98.0
> 56	57	2.0	100.0
Total	3,079	100.0	

Modes of Ventilation: Transport Ventilators

The transport of a patient, whether in the field or within the hospital, is frequently associated with physiologic instability. Alterations in gas exchange and cardiopulmonary hemodynamics are common. As a result, during periods of transport, tight control over ventilatory variables is frequently maintained.

Unfortunately, no data exist to assist in guiding the selection of mode of ventilation during transport; however, it seems reasonable to select the mode of ventilation that ensures a consistent gas delivery, yet is also able to respond to the patient. As is the case with most available transport ventilators, the mode of choice is volume assist/control (see Table 8). Although the inclusion of SIMV and pressure support seems reasonable based on usage in hospitals, the desire for consistency of mode during transport is offset by the physical constraints and costs of transport ventilators. None of the units currently available is capable of providing gas delivery in the SIMV mode consistent with that provided by ICU ventilators, although the Impact ventilator probably comes closest.[45] No transport ventilator has attempted to include pressure support as an available mode.

Modes of Ventilators: Home Care Ventilators

My observation has been that the general rule regarding maintenance of long-term, ventilator-dependent patients in the home is to maximize ventilatory muscle function to ensure the greatest periods of ventilator independence. This concept frequently differs greatly from that in the ICU, where PVS is commonly the approach used, once the most acute phase of ventilator dependence is past, with the overriding goal being complete ventilator independence. With long-term ventilatory assistance, independence is not a primary goal. In addition, in the home care setting, ventilation is generally provided to a stable cardiopulmonary system in which marked alterations in compliance and airways resistance are not a problem. As a result, the mode of ventilation that is essential on home care ventilators is volume assist/control[46] (Table 8). Although many practitioners prefer SIMV or PSV in the ICU, neither of these modes can be considered

Table 8. Modes of Ventilation for Transport and Home Care Ventilators

	Essential		Recommended		Optional	
	Transport	Home	Transport	Home	Transport	Home
Volume Assist/Control	Yes	Yes	—	—	—	—
Volume SIMV	No	No	Yes	No	No	
Pressure Support	No	No	No	No	No	No
BiPAP	No	No	No	Yes	No	—
Pressure Control	No	No	No	No	No	Yes
HMV Pressure or Volume	No	No	No	No	No	No
APRV	No	No	No	No	No	No

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*SIMV = synchronized intermittent mandatory ventilation; BiPAP = bilevel positive airway pressure; MMV = mandatory minute ventilation; and APRV = airway pressure release ventilation.

an essential mode for home care ventilators. In the home, no practitioner is available to provide the frequent attention to details of ventilation required with SIMV and PSV. Most importantly, the gas delivery system of home care ventilators does not allow for an acceptable imposed work load during the spontaneous phase of SIMV.[15] All home care ventilators impose high work loads during SIMV, and because of size and cost constraints it is doubtful that technical modifications that allow acceptable work loads will be forthcoming in the near future. Pressure support, because it is by definition a pure assist mode of ventilation, cannot be considered essential or recommended for home care ventilators. However, the use of pressure-limited ventilation, as in BiPAP, for noninvasive ventilation has demonstrated remarkable success.[41,42] It is reasonable to expect that this approach to ventilation would perform appropriately in patients with artificial airways, provided alarms are included. As noted in Table 1, BiPAP is pressure-targeted A/C or SIMV.

PEEP/CPAP and F_{IO_2}

The primary ventilator variables associated with the maintenance of oxygenation are PEEP, CPAP, and fractional concentration of inspired oxygen (F_{IO_2}) (Table 9). As a result, it is difficult to argue against the inclusion of the full spectrum of F_{IO_2} and PEEP/CPAP capabilities on all ICU ventilators (that is, F_{IO_2}s from 0.21 to 1.0 must be available). The maximum level of PEEP and CPAP to be included is debatable, although, in my experience, it is extremely rare that greater than 20 cm H_2O PEEP and 15 cm H_2O CPAP are ever applied. However, to accommodate the desires of all clinicians, both PEEP and CPAP should be available to 30 cm H_2O.

During transport, particularly intrahospital, high F_{IO_2}s (1.0) are generally the rule because of the frequency of transport-related instability.[1] In addition, the same level of PEEP considered essential with the ICU ventilator (30 cm H_2O) must be considered essential with transport units to ensure alveolar stability during transport. However, CPAP is not considered essential or even recommended on transport ventilators. In addition, none of the transport units currently available can accommodate spontaneous ventilation, particularly in association with CPAP, without imposing excessive work loads.

With home care ventilators, the tradeoffs with F_{IO_2} and PEEP/CPAP are clinical necessity vs cost, complexity, and physical size limitations. Because patients being maintained at home on mechanical ventilators are, by definition, chronically (not acutely) ill, the need for high and precise F_{IO_2} is limited, and the need for PEEP/CPAP rare. In addition, portability is a primary concern. The provision of high and precise F_{IO_2} demands a high-pressure gas source of large flow capability, which greatly limits portability. The present methodologies[47] for enhancing F_{IO_2} (gas accumulators) generally provide the range (up to 35-45%) and the accuracy (± 5%) required in the stable long-term, ventilator-dependent patient.

It is rare that home care patients require PEEP/CPAP.[46] As already indicated, all of these patients

Table 9. Suggestions for PEEP, CPAP, F_{IO_2}, and Humidification-System Features for ICU, Transport, and Home Care Ventilators

	Essential			Recommended			Optional		
	ICU	Transport	Home	ICU	Transport	Home	ICU	Transport	Home
F_{IO_2}	≤ 1.0	≤ 1.0	0.21	—	—	0.21-0.40	—	—	≥ 0.40
PEEP (cm H_2O)	≤ 30	≤ 30	No	—	—	No	—	—	≤ 10
CPAP (cm H_2O)	≤ 30	No	No	—	No	No	—	No	No
Humidifier	Yes	No	No	—	No	Yes	—	Yes	—

are stable, although chronically ill. The present design of home care ventilators increases the probability that PEEP or CPAP will greatly increase the imposed work of spontaneous breathing; this includes triggering of volume A/C because none is PEEP-compensated. As a result, sensitivity must be carefully set during A/C. If attempts are made to provide PEEP during SIMV or CPAP, dramatic increases in imposed WOB occur because no mechanism is available to prevent the patient from needing to decompress the PEEP/CPAP in order to inspire gas from the piston chamber or the environment.[15] Unless dramatic changes are made in the design of home care units, and more acutely ill patients are discharged home, the inclusion of PEEP/CPAP must be considered optional.

Humidification

With any long-term approach to ventilatory support, adequate humidification is essential to ensure appropriate function of the mucociliary escalator and to enhance secretion mobilization. With all ICU and home care ventilators, humidification systems are essential; however, with transport units they are not. During transport, the use of a humidifier frequently complicates the setup, and because ventilation is only provided for a short period with these units, no humidifier is generally the rule, not the exception. During transport outside the home, heat and moisture exchangers are commonly used.[46] To facilitate movement and mounting on wheelchairs, the humidifier should not be an integral part of the ventilator, although it does need to be available for use with all home care ventilators.

Tidal Volume and Rate

All mechanical ventilators must have the ability to specify the delivery of precise V_T and rate (f). Although no data exist to specify rate range, if one considers the age range across which ventilators are commonly used, a maximum rate for ICU ventilators and transport ventilators of 60/min and 40/min for home care units seems appropriate. The need to exceed these rate limits is rarely present even in patients markedly stressed or with large minute volume demands who require small V_Ts. The V_T delivery range on most ventilators is 0.1-2.0 L. This level has been chosen to ensure that the ventilator can accommodate the demands of the large, markedly stressed adult and to allow standard ventilators to operate pneumobelts,[47] as well as to ventilate patients with cuffless tracheostomy tubes or large bronchopleural fistulas. This upper limit of V_T seems reasonable for standard ventilators of all categories. On the lower end of the spectrum, a V_T of 100 mL seems an acceptable minimal setting because the basic ventilator in all classes is generally designed to ventilate pediatric to adult patients.

Flowrate and Waveform

At the present time (1992), numerous gas delivery waveforms are available on mechanical ventilators. Historically, waveforms have fit into four categories: square, sine, decelerating, and accelerating; although the Hamilton Veolar allows for the delivery of a total of seven different waveforms, the additional three are modifications of the standard four. However, justification for the use of various waveforms under specific clinical settings is lacking. I believe that studies attempting to establish physiologic effects of various waveforms and their clinical efficacy have failed to clearly define differences.[27,33,48-50] Specifically, no discernible differences have been noted between square and sine waveforms. In addition, no scientific data are available to establish the need to use an accelerating waveform; to my knowledge, no author has defined the need to include this waveform on any ventilator. The one waveform for which at least minimal data suggest better gas distribution and gas exchange in the presence of $\dot{V}/\dot{Q}$ abnormalities is the decelerating-flow waveform.[27,33,48] As a result, I believe that sine or square waveforms are essential, with the decelerating waveform recommended, and all others unnecessary.

Peak inspiratory flow should meet the patient's peak inspiratory flow demands.[51,52] Of course, this is not possible under every condition because adults with normal pulmonary mechanics can generate peak flows of 300 to 500 L/min. However, ICU and transport ventilators must be able to meet the spontaneous peak flow demands of patients in acute ventilatory failure. In general, this requires peak flows of up to about 120 L/min, although peak flow capability to 180 L/min is preferred, especially in CPAP and pressure support. With home care ventilators, peak inspiratory flow demands are less than with ICU and transport ventilators. Peak flows of 100 L/min generally meet the inspiratory demands of all stable chronically ventilated patients.

Inspiratory Time and I:E

As with many of the other variables discussed, little specific information is available to establish essential guidelines regarding inspiratory time and I:E. Information in the mechanical ventilation literature on the advantages of lengthened inspiratory time is controversial.[20,21,23-33] Retrospective uncontrolled data suggest that lengthy inspiratory times with inverted I:E enhance gas exchange.[23-26] However, recent data indicate that there is no benefit from increasing inspiratory time or inverting I:E if total PEEP or mean airway pressures are held constant.[27-31] As a result, the capability of achieving an I:E of 1:1 is essential, whereas inversion of the I:E is considered an option. In light of this, an inspiratory time of a maximum of 3 seconds in ICU ventilators is recommended (that is, the ability to achieve an I:E of 1:1 at a rate of 10/min). In both transport and home care ventilators, a maximum I:E of 1:1 is also recommended, with an inspiratory time of up to 2 seconds.

Maximum System Pressure

The maximum airway pressure allowable by mechanical ventilators has never been specifically addressed. Some authors[32,33,53,54] have proposed limiting peak alveolar (plateau) pressure to 40-45 cm H_2O in order to minimize the potential for all types of barotrauma. Although I agree with these authors, the specific limit that should be placed on peak airway pressure during VCV, PSV, and PCV is difficult to define. Most of today's ICU ventilators allow peak airway pressures of about 100 cm H_2O. When one considers the potential difficulties encountered in ventilating patients with high airways resistance and the lack of data addressing peak airway pressure limitations, 100 cm H_2O appears to be the maximum peak airway pressure necessary on the basic ICU ventilator and on transport ventilators. Because home care patients are stable and do not require high airway pressures, I propose a maximum peak airway pressure of 80 cm H_2O.

During pressure-limited approaches to ventilation on ICU ventilators, I would arbitrarily limit maximum pressure to 60 cm H_2O. Because all pressure-limited approaches incorporate decelerating-flow waveforms, peak inspiratory pressure closely approximates peak alveolar pressure. Considering the data reviewed by Marini et al,[32,33] Gattinoni et al,[53] and Hickling et al[54] regarding barotrauma, 60 cm H_2O appears to be a reasonable, safe upper limit for the basic ICU ventilator.

Future Prospects

It is obvious to the reader that the majority of the conclusions I have drawn regarding minimal essential gas delivery features of mechanical ventilators are based on my clinical experience and my interpretation of the literature (Table 10). However,

Table 10. Minimal Essential Ranges for Ventilator Variables

	ICU	Transport	Home
Flow (waveform)	Square or sine	Square or sine	Square or sine
Flowrate (L/min)	20-120	20-120	20-100
V_T (L)*	0.1-2.0	0.1-2.0	0.1-2.0
Inspiratory time (s)	≤ 3	≤ 2	≤ 2
I:E	≤ 1:1	≤ 1:1	≤ 1:1
Pressure Volume (cm H_2O)	1-100	1-100	1-80
Pressure PSV (cm H_2O)	1-60	—	—
Rate (per min)	1-60	1-60	1-40
PEEP (cm H_2O)	1-30	1-30	—

* V_T = tidal volume; I:E = inspiratory-expiratory time ratio; PSV = pressure support ventilation; and PEEP = positive end-expiratory pressure.

the one conclusion I feel comfortable in making is that data are lacking to guide our description of the essential features of mechanical ventilators. My hope is that this statement, others in this issue, and the Consensus Statement will establish dialogue and foster more research in this most important aspect of respiratory care.

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Mechanical Ventilator Design and Function: The Trigger Variable

Catherine SH Sassoon MD

I. Introduction
II. Pressure-Triggering in a Demand-Flow System
III. Flow-Triggering in the Flow-By System
IV. Effects of Circuit Pressure-Sensing Sites on the Trigger Variable
V. The Trigger Variable and Inspiratory Muscle Work

Introduction

The trigger variable is defined as the variable that is manipulated to deliver inspiratory flow. Although triggering comprises only a small part of the entire inspiratory cycle, inappropriate setting or design may increase the patient's effort and inspiratory muscle work.[1] The trigger variable may be a set time, pressure, volume, or flow. Time-triggering operates when the ventilator delivers a breath according to a set frequency, independent of the patient's spontaneous effort.[2] With pressure-, volume-, and flow-triggering, the ventilator delivers a breath once the set pressure, volume, or flow sensitivity is attained, independent of the set frequency. Volume-triggering has not been commonly used and at this writing (1992) is available only in the Dräger Babylog ventilator.[2] My discussion focuses on the basic characteristics of the pressure- and flow-trigger variables of a microprocessor-based ventilator, and the extent to which these variables affect inspiratory muscle work during spontaneous and mandatory (assisted) breaths. I developed this information from working with the Puritan-Bennett 7200a ventilator (P-B. Puritan-Bennett Corp, Carlsbad CA). It is not my intention to make comparisons among the different ventilators, but, where necessary, other ventilators will be mentioned for illustrative purposes.

Pressure-Triggering in a Demand-Flow System

In a demand-flow system, the trigger variable is a set pressure that must be attained at the onset of inspiration for the ventilator to deliver fresh gas into the inspiratory circuit. Pressure sensitivity is commonly expressed with a negative sign but, to avoid possible confusion, the negative sign is omitted in this text. Hence, an increase or decrease in sensitivity means an increase or decrease in the absolute value. Most microprocessor-based ventilators use pressure-triggering to initiate both mandatory breaths (assist-control, or AC: and synchronous intermittent mandatory ventilation, or SIMV) and spontaneous breaths (continuous positive airway pressure, or CPAP; SIMV; and pressure support ventilation, or PSV).

Dr Sassoon is Associate Professor of Medicine, University of California Irvine and is associated with the Departments of Medicine, Veterans Affairs Medical Center, Long Beach, and the University of California, Irvine—California.

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Reprints: Catherine SH Sassoon MD, Pulmonary and Critical Care Section (111P), Veterans Affairs Medical Center, Long Beach CA 90822.

I will first discuss pressure-triggering during spontaneous breathing (CPAP). When the pressure waveform (measured at the proximal end of the endotracheal tube) in early inspiration is analyzed, it can be seen to consist of two parts: (A) from the onset of inspiratory effort to *measured* trigger sensitivity and (B) from *measured* trigger sensitivity to maximum pressure drop (Fig. 1).

In Part A, the patient generates an effort while the pneumatic system on the inspiratory side and the exhalation valve remain closed. The slope of Part A is determined by patient inspiratory muscle strength and drive. Part A consists of two intervals: (1) the time for pressure within the patient circuit to decline to the true trigger threshold (in this example 1 cm H_2O) and (2) the response time of the pneumatic system itself once this threshold is reached (approximately 6 ms) and the time due to other factors that may lengthen or shorten the duration of Part A. These factors include (1) errors due to the speed of the pressure signal, (2) errors due to digital sampling of the transducer, (3) errors in the pressure-transducing circuit, (4) discrepancies between set and actual positive end-expiratory pressure (PEEP), and (5) circuit noise, PEEP-loss compensatory flow, or any other ventilator-based correction routine.

Errors Due to the Speed of the Pressure Signal

Pressure signals travel at the speed of sound, approximately 1 foot/ms at sea level. The pressure transducer that senses the set sensitivity for flow output within the ventilator is separated from the

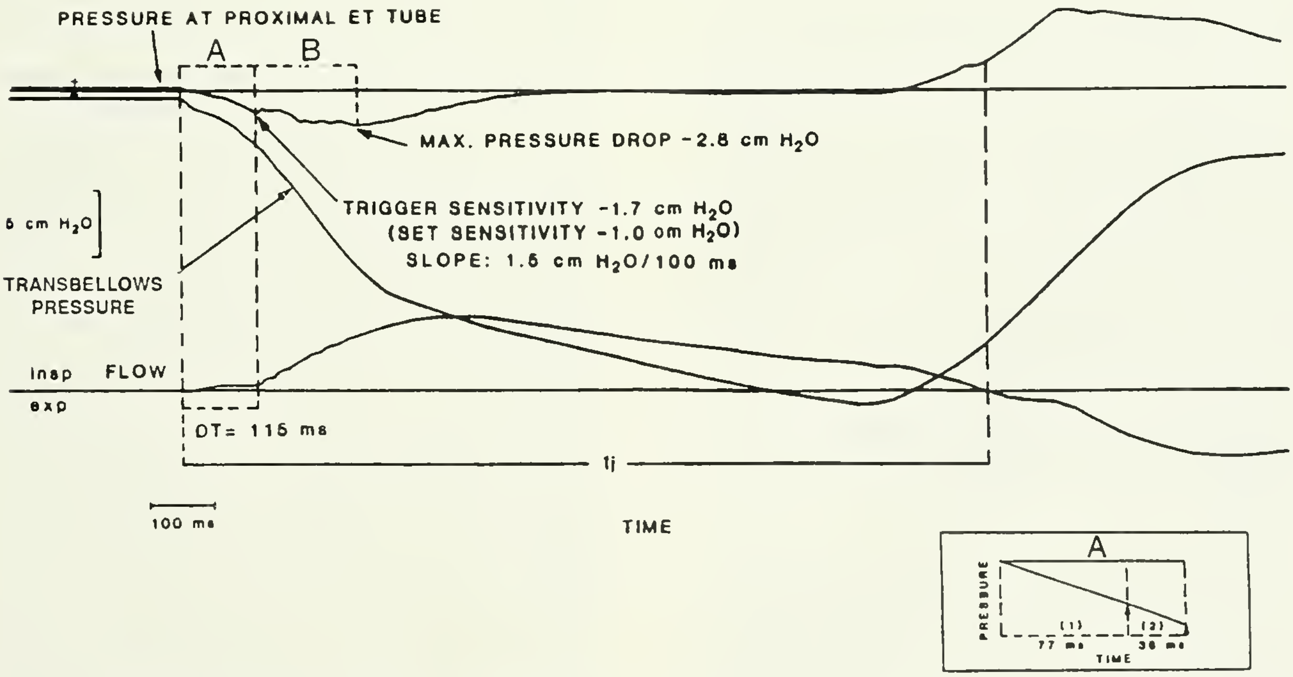

Fig. 1. Pressure-triggered or demand-flow system applied to a mechanical lung model. Pressure is measured at the proximal end of the endotracheal tube (7.5 mm), and transbellows pressure and flow tracings are generated using a one-compartment computer-driven mechanical lung model (tidal volume of 500 mL, a rapid ascending and gradual descending ramp flow waveform at a rate of 45 L/min, and compliance of 25 mL/cm H_2O) ventilated with a demand-flow CPAP system set at a CPAP level of 0 cm H_2O without a humidifier (Puritan-Bennett 7200a). Part A, trigger phase = from onset of inspiratory effort to onset of flow delivery. Flow is slightly detectable during Part A as a result of circuit length and compliance. Part B, post-trigger phase = from measured trigger sensitivity to maximum pressure drop. Inset: A-1 is from onset of inspiratory effort to set sensitivity or trigger threshold; A-2 is from trigger threshold to onset of inspiratory flow. See text for further explanation. CPAP = continuous positive airway pressure; DT = time delay from onset of inspiratory effort (negative deflection of transbellows pressure) to onset of flow; t_i = inspiratory time.

patient Y (at which pressure is recorded) by 60 inches of tubing, while the patient Y is 60 inches from the pneumatic system. Thus, it will take a minimum of a 10-ms delay from the time the pneumatic valve is signaled to open until the just-initiated flow reaches the patient Y.

Errors Due to Digital Sampling of the Pressure Transducer

Microprocessor-based systems, by design, operate in discrete time rather than continuous time. This means that the pressure transducer measuring patient-circuit pressure is polled every X ms, rather than continuously. The average increase to Part A (ie, the polling time) is therefore X/2 ms. For example, the 20-ms sampling time in the P-B 7200a increases the duration of Part A an average of 10 ms.

Errors in the Pressure-Transducing Circuit

Transducers of the quality found in life support devices exhibit an error described by the term ± (0.1 + 3.0 % of reading in cm H_2O) according to a 1991 personal communication from Warren Sanborn PhD, Puritan-Bennett. Thus, when PEEP is zero, the pressure-sensing transducer exhibits an error of ± [0.1 +(3.0 · 0)], or 0.1 cm H_2O. A minus error shortens and a plus error lengthens Part A.

Errors Due to Discrepancies between Set and Actual Positive End-Expiratory Pressure (PEEP)

The presence of intrinsic PEEP (auto-PEEP) increases the sensitivity relative to the set sensitivity, causing Part A to lengthen. Conversely, with a set PEEP value, leaks in the patient circuit decrease the actual sensitivity relative to the set sensitivity, shortening Part A.

Errors Due to Noise in the Patient Circuit

Such noise includes PEEP-loss compensatory flow or any other ventilator correction-based routine aimed at enhancing transducer sensitivity or accuracy. The baseline pressure signal (as 'seen' by the pressure transducer) is rarely 'clean,' or free of noise. The addition of signal filtering increases the time required to verify that an apparent trigger signal is a true trigger signal. When a PEEP value is selected, patient-circuit pressure declines when circuit leaks occur. If the decline in pressure equals the trigger threshold, the ventilator autocycles. Correction for those problems usually entails increasing the magnitude of the sensitivity setting, which lengthens Part A. Attempts to design a correction feature into the ventilator's operation may yield measured success, but usually at the expense of lengthening Part A.

The time components of pressure-triggering are illustrated in Figure 1. I obtained the tracings from a mechanical lung model ventilated with demand-flow CPAP. If we consider a perfect system with a set sensitivity of 1 cm H_2O, a CPAP level of 0 cm H_2O, and a slope of 1.5 cm H_2O/100 ms during Part A, 67 ms must elapse for pressure at the proximal end of the endotracheal tube (at the Y) to decline 1 cm H_2O. Ten additional milliseconds must be added to account for the speed of the pressure signal (as described earlier). Thus, even with a perfect system, a delay time of 77 ms is required before flow delivery occurs. Realistic additional delays include an average of 10 ms due to transducer polling (range 0-20 ms), 6.7 ms due to an allowable 0.1-cm H_2O error in the pressure transducer, and 6 ms due to the response time of the proportional solenoid valve, for a total of 22.7-32.7 ms (considering a maximum polling time). As shown in Figure 1, for a total delay time of 115 ms from the onset of inspiratory effort to the onset of flow measured at the Y, the additional errors account for 38 ms (Fig. 1, inset). This suggests that the entire pneumatic system still experiences unexplained delays of 5.3-15.3 ms.

Once flow begins, pressure measured at the Y continues to decline (Part B). Although Part B is no longer part of the trigger variable, it is essential to discuss it within this context to be able to distinguish differences in inspiratory muscle work imposed by pressure- versus flow-triggered CPAP systems (discussed later). The continued decline in pressure in the post-trigger phase (Part B) is explained by insufficient initial flow delivery, and is a function of the ventilator flow-pressure control algorithm. The change between the pressure within the circuit (as a result of the triggering) and a target pressure is the feedback variable most commonly used to control flow and pressure. This target pressure in the demand-flow system of the P-B 7200a is slightly below the end-expiratory airway pressure (discussed later). The pressure gradient between circuit and target pressures is sensed at one of these sites: inhalation port, exhalation port, or Y.

The delivery of insufficient initial flow may result in an excessive airway pressure drop (Fig. 1, Part B) and can result in increased inspiratory muscle work.[3,4] A demand-flow CPAP system with an inadequate delivery of flow in early inspiration is shown in Figure 1. At 0 cm H_2O CPAP, pressure measured at the proximal end of the endotracheal tube remains below atmospheric pressure and increases slightly above atmospheric pressure near the end of inspiration. I believe that this phenomenon (ie, the insufficient flow) could be 'tuned out' if it were possible to adjust flow gain. However, it remains to be determined whether an adjustable flow gain tailored to the patient's ventilatory demand would reduce inspiratory muscle work significantly.

During volume-limited (eg, AC, SIMV) or pressure-limited (eg, PSV) breaths, the trigger phase (Part A) is similar to that during CPAP. In the post-trigger phase, alteration of the peak inspiratory flowrates[5,6] or adjustment of initial flow delivery[7,8] remain essential elements in meeting the patient's early demand for flow and determining inspiratory muscle work.

Flow-Triggering in the Flow-By System

Flow-triggering, also referred to as Flow-By, is currently available for CPAP and SIMV (during both spontaneous and mandatory breaths) only in the P-B 7200a ventilator. I believe that flow-triggering will soon be available for all breaths, including both volume- and pressure-limited breaths. The flow-by system consists of two set variables, the base flow and the flow sensitivity. (Flow-By is the trademark of an option available on the P-B 7200 ventilator; hereafter in this paper flow-by will be used generically.) The base flow can be set between 5 and 20 L/min, while the flow sensitivity can be set from as low as 1 L/min to a maximum of one half of the base flow value. The base flow consists of fresh gas that continuously circulates in the inhalation and exhalation circuits, which may result in a slight PEEP. This base flow exits through the exhalation port and is measured every 20 ms. The patient's earliest demand for flow is satisfied by the base flow. The flow sensitivity is computed as the difference between the base flow and the exhaled flow. Thus, flow sensitivity is the magnitude of the flow (diverted from the exhalation circuit into the patient's lungs) that causes the ventilator to trigger. As the subject inhales, and the flow sensitivity is reached within a specific 20-ms interval, the flow-pressure control algorithm is activated, the pneumatic valve opens, and fresh gas is delivered. As with pressure-triggered demand-flow, fresh gas at a flowrate up to 180 L/min can be supplied, if needed, to maintain the airway pressure at the preset CPAP level. During the trigger phase (Fig. 2, Part C), the exhalation valve remains partially open, but at the onset of gas delivery the exhalation valve either closes or remains partially open, depending on the magnitude of the patient's inspiratory effort. A vigorous inspiratory effort effectively closes the exhalation valve.

Differences between flow-by and demand-flow systems can be examined within the trigger and post-trigger phases. Differences within the trigger phase are primarily related to the time delay in opening the pneumatic system. The relationship among measured trigger sensitivity, time delay from the onset of inspiratory effort to the onset of flow delivery, and set trigger sensitivity for both flow-by and demand-flow systems using a mechanical lung model is illustrated in Figure 3A. The measured trigger sensitivity is the pressure from the end-expiratory pressure to the lowest level during the trigger phase. Note that on flow-by, the flow sensitivity (and not the measured trigger sensitivity) triggers the pneumatic valve to open. The time delay from the onset of inspiratory effort to the onset of flow at the minimum set sensitivity with flow-by of 1 L/min is only slightly less than at the minimum set sensitivity with demand flow of 0.5 cm H_2O (65 vs 75 ms, respectively) (Fig. 3A). However, at the set sensitivity that is commonly used in clinical practice of 2 L/min on flow-by and 1 cm H_2O on demand-flow, the time delay is 75 ms vs 115 ms, respectively (Fig. 3A). More importantly, for a given time delay (eg, 75 ms), flow-by re-

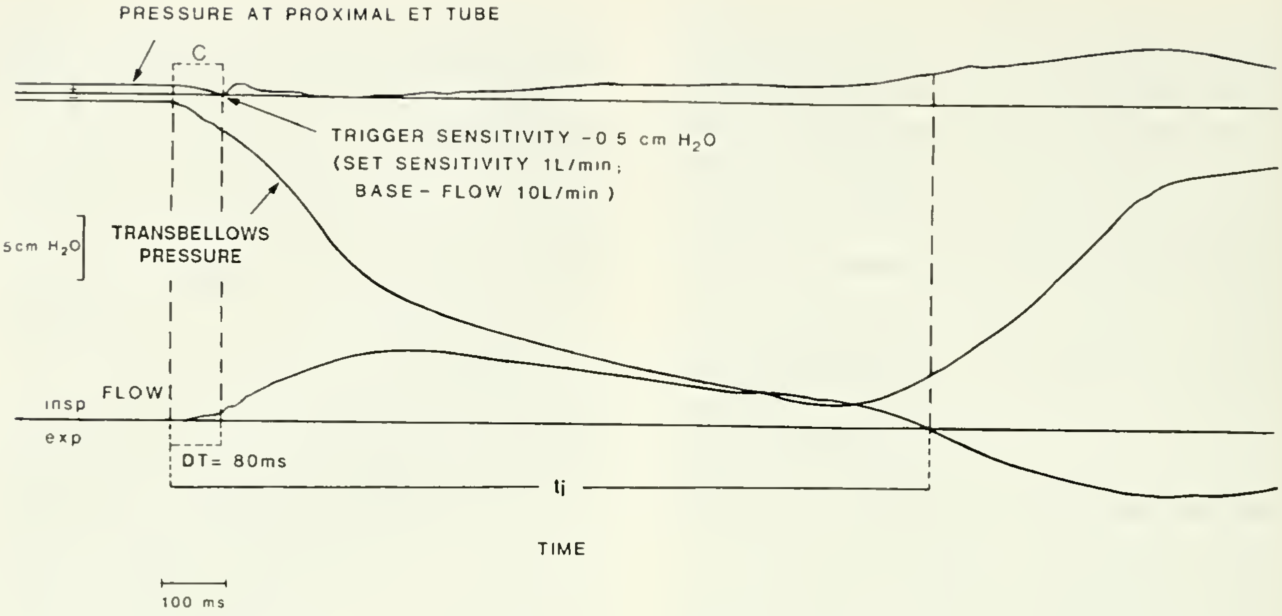

Fig. 2. Flow-triggered or flow-by system applied to a mechanical lung model at a CPAP level of 0 cm H_2O. Settings of mechanical lung model are the same as in Figure 1. Part C = from inspiratory effort to opening of the pneumatic system. Flow during Part C is provided by the base flow. Note: Airway pressure remains at or above atmospheric pressure, acting as a small inspiratory pressure-assist due to the relatively optimal flow-control algorithm. The measured trigger sensitivity of 0.5 cm H_2O is not the actual sensitivity that triggers the pneumatic system to open. See Figure 1 for definition of abbreviations.

quires a set sensitivity of 2 L/min while demand-flow requires 0.5 cm H_2O. The latter is a very sensitive setting that may cause autocycling.

During the trigger phase, the effect of flow- and pressure-triggering on the pressure-time product is essentially unknown. Because, during the trigger phase, changes in lung volume are negligible with both flow- and pressure-triggering, the pressure-time product (an estimate of the oxygen consumption of the inspiratory muscles[9-11]) would be an appropriate parameter to measure. Substituting measured trigger sensitivity in Figure 3 with changes in transbellows pressure (ΔP_{tb}), the transbellows pressure-time product during the trigger phase can be assessed (Fig. 3B). ΔP_{tb} is the change in P_{tb} from the onset of inspiratory effort to the onset of flow delivery. The slopes of the relationship between ΔP_{tb} and the time delay for both flow- and pressure-triggering are not significantly different. Hence, for a given time delay, the ΔP_{tb}-time product of both flow- and pressure-triggering is similar. Because the total time delay with flow-triggering is relatively short, the pressure-time product during the trigger phase would be less than with pressure-triggering. However, with the improved design of microprocessor-based ventilators and a relatively sensitive trigger setting used in clinical practice, the effect of flow- or pressure-triggering on the pressure-time product is probably small. Yet, a prolonged time delay with pressure-triggering may result in patient discomfort and dyspnea.[12,13]

Because of the current design of the flow-pressure control algorithm, flow-by is more responsive to patient ventilatory demand during the post-trigger phase than is demand-flow. Immediately after the trigger sensitivity is attained, airway pressure increases and is maintained above atmospheric pressure throughout the inspiratory cycle, acting as a small inspiratory pressure-assist (Fig. 2). Once triggering has occurred, as with demand-flow, the gradient between the pressure within the circuit and a target pressure, rather than flow, re-

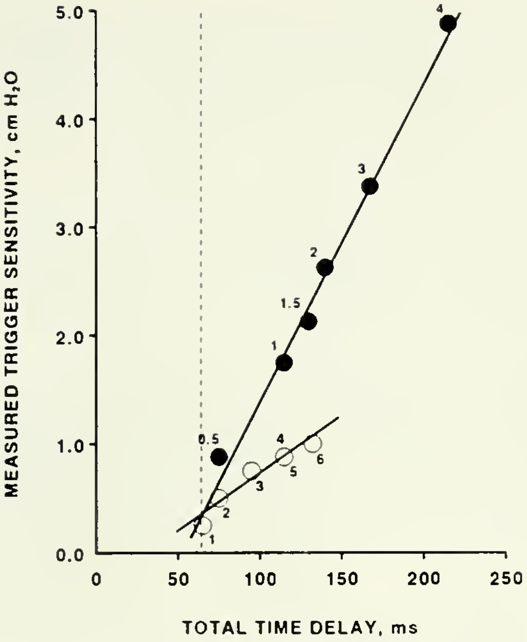

Fig. 3A. The relationship between measured trigger sensitivity (pressure drop at the Y just before the opening of the pneumatic system), total delay time from the onset of inspiratory effort to the onset of flow delivery, and set trigger sensitivity for both demand-flow (●) and flow-by (○) using a mechanical lung model (tidal volume 500 mL, a gradual ascending and descending ramp flow at 40 L/min, and compliance 25 mL/cm H_2O, with an endotracheal tube size 9.0 mm). Numbers next to symbols are the set sensitivity in cm H_2O for demand-flow and L/min for flow-by. It should be noted that the measured trigger sensitivity (cm H_2O) for flow-by is not the sensitivity that triggers the pneumatic system to open. The dashed line is the line passing through the minimum delay time (65 ms) of set flow sensitivity of 1 L/min. For a demand-flow system, for the same minimum delay time, pressure sensitivity has to be set at < 0.5 cm H_2O. The latter exceeds the sensitivity limit of the ventilator.

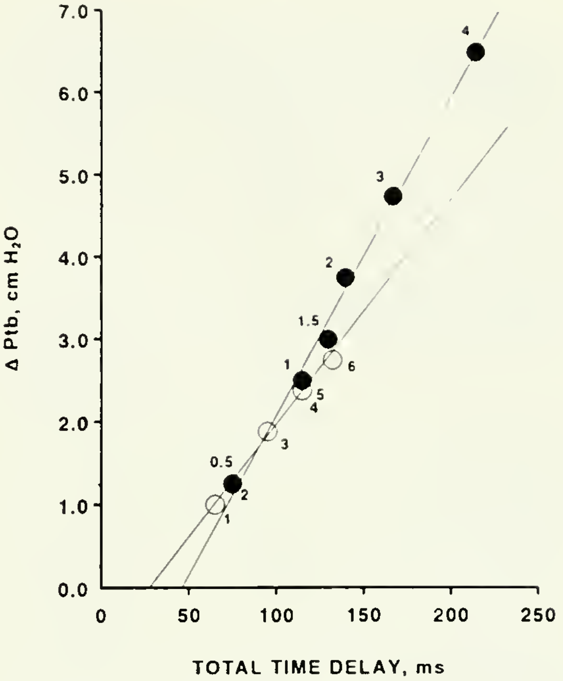

Fig. 3B. The relationship between changes in trans-bellows pressure (ΔP_{tb}) and the total delay time during the trigger phase. In Figure 3, ΔP_{tb} is substituted for measured trigger sensitivity. ΔP_{tb} is the change in trans-bellows pressure from the onset of inspiratory effort to the onset of flow delivery. The slope of the relationship for demand-flow (●) is not significantly different from that for flow-by (○). $Y = 0.04X - 1.77$ for demand-flow, and $Y = 0.03X - 0.75$ for flow-by.

mains the feedback parameter sent to the ventilator. In flow-by, this target pressure is slightly above the end-expiratory airway pressure. Figure 2 demonstrates the pressure tracing measured at the Y of a mechanical lung model ventilated with a flow-by CPAP of 0 cm H_2O, set base flow of 10 L/min, and flow sensitivity of 1 L/min. In the immediate post-trigger phase, pressure increases and is maintained above atmospheric pressure throughout inspiration, suggesting the adequate response of the flow-pressure control algorithm in comparison to demand-flow (Fig. 1). Differences in the post-trigger phase account for increases in inspiratory muscle work imposed by demand-flow.[3,4] The functional characteristics of demand-flow and flow-by systems are summarized in Table 1.

Effects of Circuit Pressure-Sensing Sites on the Trigger Variable

Conventionally, pressure in the patient circuit is sensed in one of three locations to determine whether circuit pressure has reached the trigger threshold: the exhalation port (P-B 7200a, IRISA, Siemens 900C), the inhalation port (typically 'inside' the ventilator, Hamilton Veolar), or the patient Y (Bear 5). Wherever pressure is sensed, the physical location of the pressure transducer is within the ventilator's electronics.

During the trigger phase with demand-flow, the patient circuit is a closed system (ie, the pneumatic

Table 1. Characteristics of Pressure- and Flow-Trigger Variables during Spontaneous Breathing (CPAP)

	Pressure-Trigger	Flow-Trigger
Set Sensitivity	Pressure	Flow
Trigger Phase:		
Exhalation valve	Closed	Open
Inspiratory flow	Negligible due to circuit compliance	Provided by base flow
Time delay	Relatively long	Relatively short
Post-Trigger Phase:		
Exhalation valve	Closed	Open or closed depending on patient effort
Feedback signal for flow-pressure control	Circuit pressure	Circuit pressure
Target pressure for flow-pressure control	Below end-expiratory pressure, hence may result in under-delivery of flow (in some ventilators)	Above end-expiratory pressure, hence the relatively optimal flow delivery

system on the inspiratory side and the exhalation valve are closed). Therefore, all three locations yield an equivalent pressure signal up to the moment of gas delivery. With flow-by, triggering is based on flow sensitivity. Hence, with both systems, the location of the circuit pressure-sensing line is of no consequence during the trigger phase. Figures 4 and 5 illustrate this identical pressure-time trace during the trigger phase for both demand-flow and flow-by.

During the post-trigger phase, for both demand-flow and flow-by, the change in circuit pressure is the feedback signal to activate the flow-pressure control algorithm. Once triggering opens the pneumatic valve, circuit pressure increases either gradually or rapidly, depending on the gain of the flow controller and the impedance at the patient Y. At some finite time, a target pressure (a value set by the manufacturer or the operator, ie, a pressure support level) is reached, and flow is modulated to maintain pressure in the patient circuit within a control band around the target pressure. As mentioned earlier, in the demand-flow system of the P-B 7200a, this target pressure is slightly below baseline PEEP, whereas in the flow-by system, it is slightly above the baseline PEEP (Fig. 6). The feedback signal to the ventilator flow-pressure controller is the pressure gradient between the sensed circuit pressure and the controller's target pressure.

To gain insight into the issue of pressure-sensing and flow control, a healthy volunteer was asked to breathe through the ventilator via a mouthpiece.

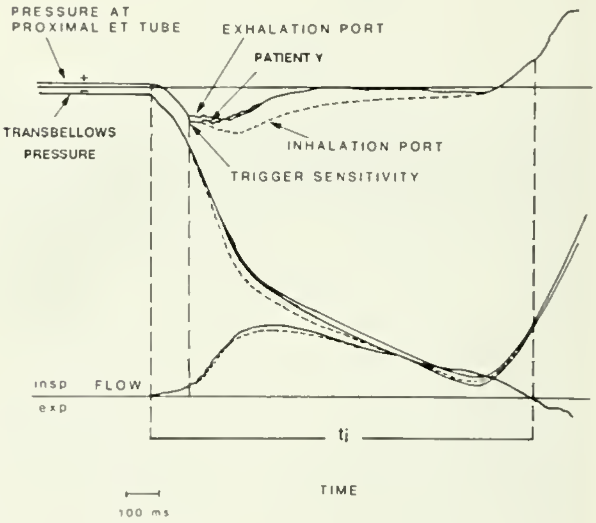

Fig. 4. Effect of circuit pressure-sensing sites (ie, exhalation port, inhalation port, and patient Y) on pressure drop measured at the proximal end of the endotracheal tube with the demand-flow CPAP system (mechanical lung model). Settings of lung model are the same as in Figure 1. Superimposed pressure tracings are pressures of three separate breaths measured with a pressure transducer at the Y. During the trigger phase, pressures are identical for all three locations (exhalation port, inhalation port, and patient Y). During the post-trigger phase, a marked pressure difference is observed between that labeled inhalation port and the exhalation port or patient Y. Pressures labeled exhalation port and patient Y reflect actual pressure within the circuit sensed by the ventilator. This is because the ventilator algorithm (P-B 7200a) is designed to sense pressure at the exhalation port. Conversely, pressure labeled inhalation port is not the actual pressure sensed by the ventilator. See text for detailed explanation. See Figure 1 for definition of abbreviations.

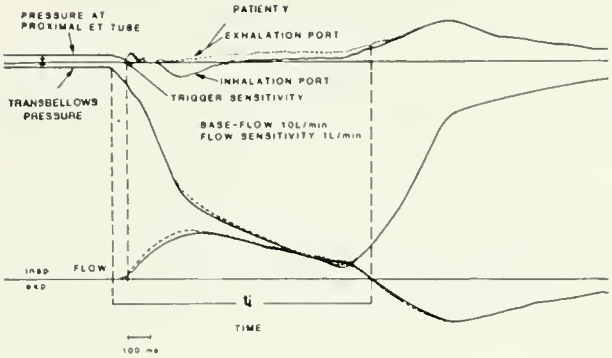

Fig. 5. Effect of circuit pressure-sensing sites on pressure drop measured at the proximal end of the endotracheal tube on flow-by CPAP system. Note that the trigger sensitivity indicated by the arrow marks only the degree of pressure drop and not the actual sensitivity that triggers the pneumatic system to open. See Figure 4 and text for further explanation.

Circuit pressures were measured simultaneously at the exhalation port (A), the inhalation port (B), and the patient Y (C). Flow was measured at the Y with a pneumotachograph while the subject was breathing quietly, first on demand-flow, and then on flow-by (Fig. 6). Note that the P-B 7200a senses circuit pressure at the exhalation port. With respect to the demand-flow breath, there are minor differences between the A and C pressure tracings, while B exhibits major differences compared to A and C. These differences are readily explained by examining the physics of flow delivery and pressure measurement relative to the patient circuit. Before the triggering of the pneumatic system, the patient circuit was closed. As the subject inhaled, pressure within the circuit decreased uniformly. However, once flow delivery began, a pressure gradient became established across the inspiratory circuit due to the resistance of the circuit. Because the exhalation circuit experienced no such flow, pressure should have remained equal throughout that tubular volume. Separation of A and C by a filter barrier is inconsequential during triggering and inspiration because the closed exhalation circuit transmits only pressure and not flow. A partially occluded filter is an impediment only to exhalation. The pressure gradient between B and C is just the amount necessary to drive the flow into the subject's lungs.

The traces seen for the flow-by breath are explained by similar analysis. With this configuration, the equivalence of the A and C traces is influenced by the aggressiveness of the inspiratory effort. During flow-triggering the exhalation valve remains open. Even after triggering, excess flow can escape through the valve. Thus, Traces A and C may exhibit minor differences, depending on the delivered flow and the subject's inspiratory effort; but, as with the demand-flow example, the pressure increase noted in Trace B compared to C represents the pressure gradient required to drive the flow into the subject's lung.

Before interpreting the relevance of the sensing location to flow delivery, consider a more controlled experiment (Figs. 4 & 5). For these studies, spontaneous breathing was accomplished with a computer-driven mechanical lung. The pressure-sensing site could be moved on successive breaths because every breath is identical. As before, flow was measured at the Y. Figure 4 is a composite of three superimposed breaths separately recorded during demand-flow trials. Pressure was sensed at each of the three locations. The upper three traces show the pressure measured at the proximal end of the endotracheal tube when sensing was conducted at the three different locations. Also shown in the figure are the flow and the transbellows pressure traces.

Analysis of the three superimposed traces illustrates the issues surrounding the pressure-sensing site relative to concurrently observed inspiratory muscle work. Again with respect to the trigger phase, all three pressure-sensing sites yield equivalent pressure traces measured at the Y.

During the post-trigger phase, pressure at the Y is modestly to markedly affected by the choice of the sensing site. Whether the sensing site is at the patient Y (C) or exhalation port (A), the transbellows pressure curves exhibit little difference. Placing the sensing site at the inhalation port (B) affects both pressures at the patient Y and transbellows pressure. Explanation of these discrepancies follows from the design of the flow-pressure control algorithm. With pressure-sensing at B, a smaller pressure gradient between circuit pressure and target pressure is fed back to the ventilator (Fig. 6). This results in an underdelivery of flow; transbellows pressure becomes more negative, suggesting that relatively more work is required to sustain inspiration. When this same protocol is re-

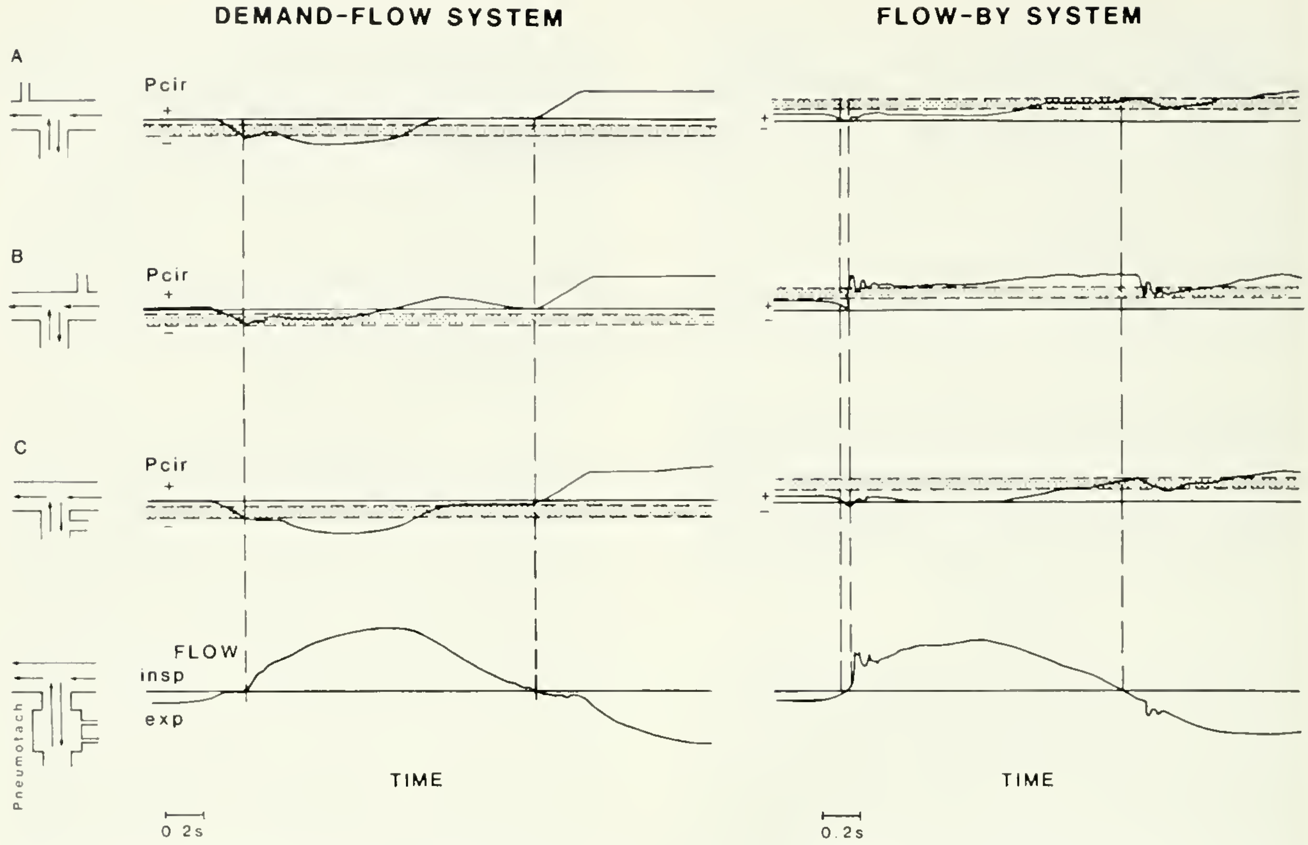

Fig. 6. Simultaneous measurements of pressures within the circuit using three pressure transducers placed at the exhalation port (A), inhalation port (B), and patient Y (C) during demand-flow and flow-by CPAP. These data were obtained by having a normal subject breathe quietly through a mouthpiece at a CPAP level of 0 cm H_2O. A pneumotachograph was placed at the patient Y for measurement of flow. Note that the pressure-sensing location remains at the exhalation port. The pressure gradient between circuit pressure (P_{cir}) and the target pressure band (shaded area) is small when measured at the inhalation port (B). This pressure gradient represents the feedback signal to activate the flow-pressure control algorithm if the pressure-sensing line were located in the inhalation port. Notice that P_{cir} may overshoot target pressure with both demand-flow and flow-by because of the inherently imperfect controller. See text for further explanation.

peated for the flow-by system, the results are qualitatively equivalent.

In modern microprocessor-based ventilators, modeling of the patient circuit with an optimal flow delivery could be achieved regardless of the sensing location. What remains to be assessed are the advantages and disadvantages attributable to sensing at the three locations, which are summarized in Table 2.

Given the knowledge that pressure-sensing at the Y contains the most relevant information before gas flow leaves the patient circuit and enters the patient lungs, it must be determined whether this location provides the optimal location to measure these pressures. The good mechanical protection at the exhalation port (A) permits accurate reading of the pressures seen at the Y during inhalation, but during exhalation, expiratory pressures are underestimated at this location. The inhalation port is also mechanically well protected, but inspiratory pressures overestimate the reading at the Y. Expiratory pressures accurately reflect those read at the Y. Sensing at the patient Y reads 'optimal' pressures during inspiration and expiration, but the mechanical environment is abusive. However, with good maintenance of the patient circuit, a reasonable level of operator skill, and good knowledge of the application of pressure support, any of the three locations provides adequate ventilator performance. Alternatively, the pressure-sensing site can be in both the exhalation and inhalation ports to read accurate pressures at the Y during inspiratory and expiratory

Table 2. Advantages and Disadvantages of the Different Circuit Pressure-Sensing Sites

Advantages	Disadvantages
A. Exhalation Port	
Well-protected from mechanical abuse.	Requires protection from moisture of exhaled gas.
During inhalation, accurately reads pressure at the Y.	During exhalation, underestimates pressure at the Y.
During inhalation, increases in inspiratory- or expiratory-circuit resistance do not compromise inspiratory-flow output, except for manyfold increases.	During exhalation, increases in expiratory-circuit resistance compromise expiratory flow. Hence, system requires well-maintained expiratory filter to ensure that expiratory-circuit resistance remains low.
B. Inhalation Port	
Well-protected from mechanical abuse.	During inhalation, overestimates pressure at the Y.
Does not require protection from moisture or additional filters.	During inhalation, increases in inspiratory-circuit resistance compromise inspiratory-flow output. For example, factors such as selection of humidifier and type of patient circuit yield varying patient inspiratory effort for fixed ventilator settings; however, most of these problems can be corrected with the use of pressure support ventilation.
During exhalation, accurately reads pressure at the Y as long as the inspiratory circuit remains patent.	
During inhalation, increases in expiratory-circuit resistance do not compromise inspiratory-flow output.	
C. Patient Y	
During inhalation and exhalation, accurately reads both inspiratory and expiratory pressures.	Susceptible to mechanical abuse.
Pressure readings reflect relative condition of inspiratory and expiratory circuits.	Requires a separate pressure-sensing tube, which is prone to occlusion, blockage, and disconnection, all of which prevent sensing of patient effort.

events, respectively. This dual pressure-sensing design would provide the advantages of sensing at the Y while eliminating its disadvantages.

The Trigger Variable and Inspiratory Muscle Work

In this section, I discuss the effect of the trigger variables on inspiratory muscle work during spontaneous breathing (CPAP) and synchronous intermittent mandatory ventilation (SIMV).

Several studies have shown that during spontaneous breathing with CPAP, inspiratory muscle work (W_i) is partly determined by the CPAP system.[14,15] From the earlier discussion, it can be predicted that the excessive airway pressure drop during early inspiration (ie, post-trigger phase) reflects increased W_i during pressure-triggering or demand-flow CPAP. Indeed, this has been consistently shown in studies of healthy subjects[14] and intubated patients[4,15,16] that compared W_i on the demand-flow and continuous-flow systems (the latter is considered to be the 'gold standard' of a CPAP system). What the minimum tolerable amount of work imposed by the demand-flow system should be is unclear. Based on studies performed on mechanical lung models, most demand-flow systems of microprocessor-based ventilators do not appear to impose excessive work.[17] However, as exemplified in Figures 7 and 8, the patient ventilatory demand cannot be ignored. Figures 7 and 8 show the airway pressure (P_{aw}), esophageal pressure (P_{es}), tidal volume (V_T), and flow ($\dot{V}$) tracings of two patients without and with increased ventilatory demand, while on demand-flow CPAP, flow-by CPAP, and PSV of 5 cm H_2O at a CPAP level of 0 cm H_2O (P-B 7200a) for 15 minutes each. In a patient without a high ventilatory demand, there is a negligible difference in the P_{es} swings between the demand-flow and flow-by systems (Fig. 7). In contrast, in a patient with a high ventilatory demand, esophageal pressure swings on demand-flow double those on flow-by (Fig. 8). The application of PSV of 5 cm H_2O eliminates the post-trigger airway pressure drop seen with demand-flow, and results in a noticeable decrease in respiratory frequency. Hence, when flow-triggering is not available, the work imposed

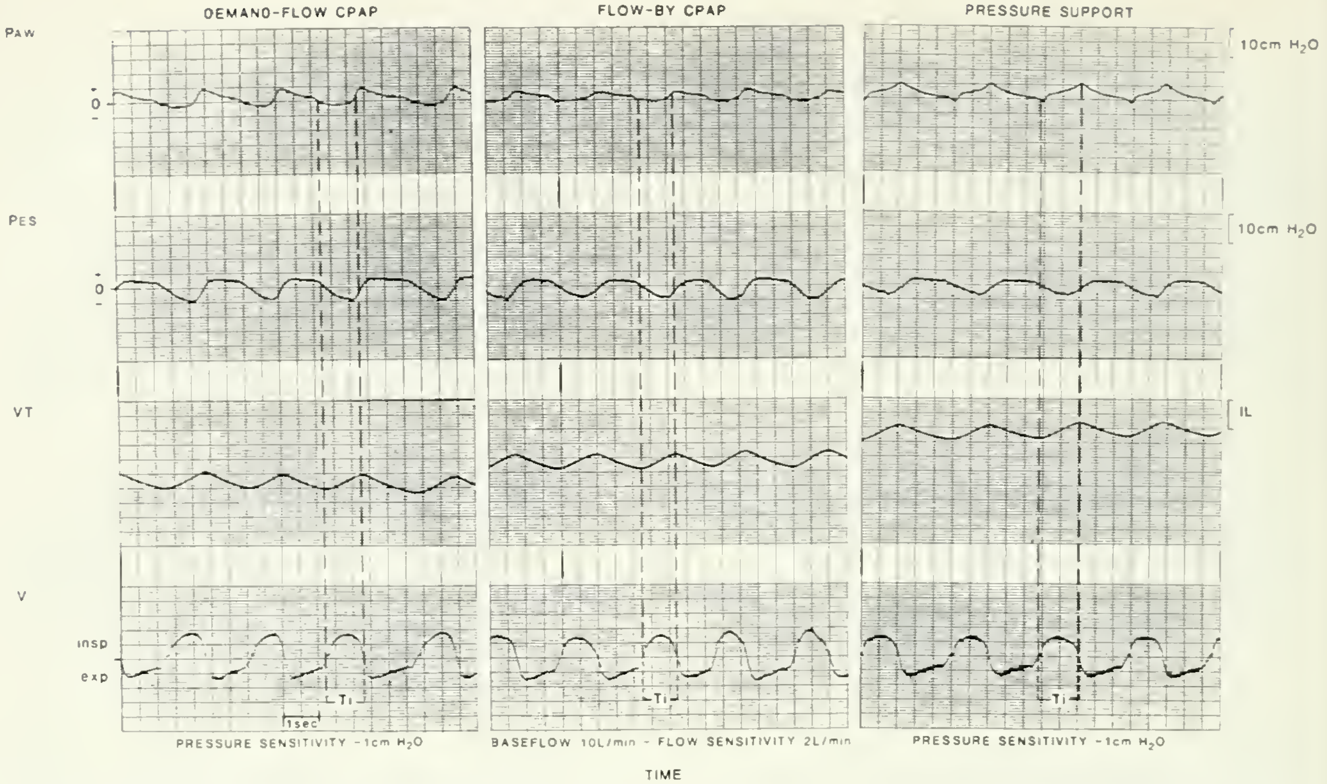

Fig. 7. Airway pressure (P_{aw}), esophageal pressure (P_{es}), tidal volume (V_T), and flow ($\dot{V}$) tracings of a patient without a high ventilatory demand on demand-flow, flow-by CPAP, and 5 cm H_2O pressure support at a CPAP level of 0 cm H_2O.

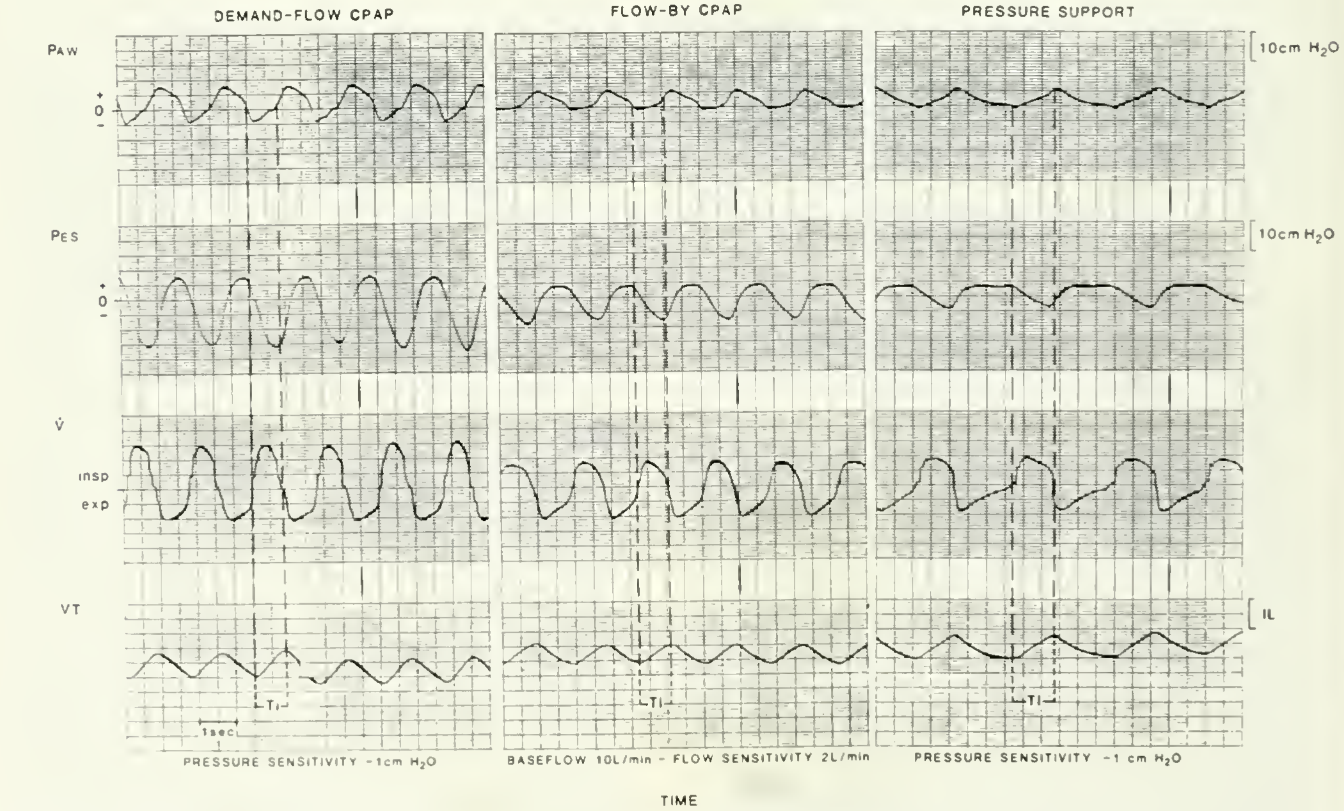

Fig. 8. Airway pressure (P_{aw}), esophageal pressure (P_{es}), flow ($\dot{V}$), and tidal volume (V_T) tracings of a patient with a high ventilatory demand on demand-flow, flow-by CPAP, and 5 cm H_2O pressure support at a CPAP level of 0 cm H_2O.

Table 3. Inspiratory Muscle Work and Maximal Airway Pressure Drop during Continuous-Flow, Flow-By, and Demand-Flow CPAP and during Pressure Support of 5 cm H_2O*

	Continuous-Flow	Flow-By	Demand-Flow	Pressure Support
W_i, J/L	0.82 (0.14)	0.69 (0.12)†	1.00 (0.16)	0.67 (0.15)†
W_i, J/min	13.24 (3.49)	10.36 (2.42)	15.87 (4.64)	10.77 (2.99)
ΔP_{aw}, cm H_2O	1.48 (0.20)‡	0.69 (0.40)†	2.79 (0.42)	1.65 (0.50)

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* Data obtained from Reference 4 (with permission) at CPAP level of 0 cm H_2O; Values are mean (SE); n = 9. Flow-by sensitivity 2 L/min, base flow 10 L/min; demand-flow sensitivity −1 cm H_2O. W_i = inspiratory muscle work, ΔP_{aw} = maximum airway pressure drop from end-expiratory pressure.

† $p < 0.01$ compared to demand-flow (analysis of variance).

‡ $p < 0.05$ compared to demand-flow (analysis of variance).

by the demand-flow system can be minimized by applying a small amount of pressure support.

W_i on the flow-by system has been shown to be comparable to W_i on continuous-flow in a mechanical lung model.[17] In a study of patients with chronic obstructive pulmonary disease who were ready to wean, my co-workers and I have recently demonstrated that W_i (J/L) and work rate (J/min) on flow-by (sensitivity 2 L/min) tends to be less than that on continuous-flow[4] and significantly less than that on demand-flow with a sensitivity of 1 cm H_2O (Table 3). On the flow-by system, the base flow values have no significant effect on W_i.[3] Differences in W_i on the various CPAP systems are not related to alterations in respiratory system mechanics, but appear to be related to the maximal airway pressure drop from end-expiratory airway pressure (Table 3). It remains to be determined whether a demand-flow system with an optimal flow-control algorithm that will maintain airway pressure at or above the end-expiratory pressure will result in a comparable degree of imposed work as with the flow-by system. Table 3 also shows that the addition of 5-cm H_2O pressure support to the demand-flow system decreases W_i to a level comparable to that with the flow-by system.

During SIMV, mandatory breaths are interspersed with spontaneous breaths. With flow-by SIMV, flow-triggering occurs during both the spontaneous and the mandatory breaths. SIMV provides an opportunity to assess the effect of the different trigger variables on W_i of the mandatory breath.[18] Unfortunately, this is not strictly the case because under SIMV conditions the preceding spontaneous breaths influence W_i of the mandatory breaths.[19] Nevertheless, at the present time, SIMV is the only mode available for such comparison.

During mandatory breaths of both flow-by and demand-flow SIMV, the extent to which the trigger variables influence W_i depends on the set sensitivity. In the post-trigger phase, peak inspiratory flowrates affect W_i.[1,5,6] In our study of patients recovering from acute respiratory failure, peak inspiratory flowrates for the mandatory breaths were set at a constant flow of 60 L/min on both flow-by and demand-flow SIMV. W_i (J/L) was calculated at various SIMV rates. At a given SIMV rate, W_i of the mandatory breath on flow-by tended to be lower than that on demand-flow, but the difference was not statistically significant (Table 4). A similar trend was found in the airway pressure drop of the mandatory breaths. As expected, W_i of the spontaneous breath was significantly less on flow-by than on demand-flow, particularly at SIMV rates of 40% and 20% of the assist/control rate. Thus, based on a small number of patients, the effect of flow- or pressure-triggering on W_i of the mandatory breath seems comparable. Conversely, during the spontaneous breath, trigger variables significantly influence W_i.

Summary

Because of the design characteristics, flow-triggering appears to offer measurable advantages over pressure-triggering, particularly during spontaneous breathing. During the trigger phase, flow-triggering provides a relatively shorter time delay than pressure-triggering. A trigger sensitivity that

Table 4. Inspiratory Muscle Work during Flow-By and Demand-Flow Synchronous Intermittent Mandatory Ventilation

	Mandatory Breath (J/L)	Spontaneous Breath (J/L)
Flow-by 60%*	0.28 (0.07)	0.36 (0.09)
Demand-flow 60%	0.37 (0.10)	0.57 (0.11)
Flow-by 40%	0.40 (0.11)	0.40 (0.10)†
Demand-flow 40%	0.46 (0.12)	0.71 (0.10)
Flow-by 20%	0.35 (0.07)	0.50 (0.10)‡
Demand-flow 20%	0.50 (0.09)	0.81 (0.12)

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* Percent of rate on assist-control (AC). Mean AC rate = 20 breaths/min. Mandatory breath settings = V_T 10 mL/kg, peak inspiratory flow 60 L/min, square-wave flow pattern. Flow-by sensitivity 2 L/min, base flow 10 L/min; demand-flow sensitivity –1 cm H_2O. Values are mean (SE), n = 8. See Table 3 for definition of abbreviations.

† $p < 0.05$ compared to demand-flow of the same rate (analysis of variance).

‡ $p < 0.01$ compared to demand-flow of the same rate (analysis of variance).

does not cause autocycling can be set while a short time delay is maintained. It remains to be determined whether flow-triggering has less effect on the pressure-time product than pressure-triggering.

During the post-trigger phase, the relatively optimal flow delivery with flow-by results in the maintenance of airway pressure at or above the end-expiratory airway pressure level. This accounts for the lower level of inspiratory muscle work observed with flow-by over that observed with demand-flow. Whether inspiratory muscle work on a demand-flow system with optimal flow delivery will be similar to that on flow-by is not known. With a flow-by or demand-flow system, the circuit pressure-sensing site influences the flow-pressure control algorithm in the post-trigger phase only. In microprocessor-based ventilators, the shortcomings seen with pressure-triggering during the post-trigger phase can unquestionably be overcome with a better ventilator algorithm design or the application of a small amount of pressure support. However, during the trigger phase, the impact of this effort is less clear.

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Inspired Gas Conditioning

Maire P Shelly MB ChB FFARCS

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Introduction

Under normal circumstances, the nose and upper airways heat and moisturise inspired gases. In certain states, such as tracheal intubation, this mechanism is inadequate and attention must be given to artificially conditioning the inspired gases. Because little is known about the mechanisms of heat and moisture exchange in the upper airway, the requirements for replacing those mechanisms is poorly understood. A variety of humidifiers is now available, but their appropriate use has not been established.

In this review, I discuss the reasons for conditioning a patient's inspired gases by examining the normal mechanisms of heat and moisture exchange and the consequences of inadequate and excessive humidification. In addition, the different types of humidifier currently available are described, and guidelines for their appropriate use are suggested. Filters are now frequently used with humidification devices; their potential value in infection control is also discussed.

Why Humidify Inspired Gases?

Inspired gases require humidification when the normal mechanisms of heat and moisture exchange are bypassed or inadequate. An examination of the normal mechanisms of humidification and the consequences of inadequate and excessive humidification should enable the requirement for heat and moisture exchange to be estimated.

Dr Shelly is Consultant in Anaesthesia and Intensive Care, University Hospital of South Manchester, West Didsbury, Manchester, United Kingdom.

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Reprints: Maire P Shelly MB ChB FFARCS, Anaesthesia and Intensive Care, University Hospital of South Manchester, Nell Lane, West Didsbury, Manchester, M20 8LR UK.

Normal Mechanisms of Humidification

Inspired air normally passes through the nose and upper airway, where it is heated to 37°C and humidified to 100% relative humidity.[1] Thus, conditions within the lower airways and alveoli remain constant, and gas exchange is efficient. On expiration, the upper airway and nose conserve heat and moisture, so losses are minimised.[2] The nose and upper airway thus act as a countercurrent heat and moisture exchanger (HME), conditioning inspired gases and retaining the heat and moisture. The point at which inspired air reaches 37°C and 100% relative humidity is called the isothermic saturation boundary.[3] This is normally just below the carina, but its position varies according to the volume, temperature, and humidity of inspired gases and the presence of a tracheal tube.[4] Above the isothermic saturation boundary, the airway acts as a heat and moisture exchanger; below it, the temperature and humidity remain constant.[1]

The upper airway is able to condition inspired gas over a wide range of conditions. In hot climates, the nose has a thermoregulatory function that depends on the humidity of inspired gases.[2] Hot, dry inspired air is cooled as it passes down the upper airway because the latent heat of vaporisation of water is taken from the mucosa. In humid climates, this does not occur, and other thermoregulatory systems must control body temperature. In cold climates, the heat and moisture requirements of inspired gases are high. To meet these requirements, the temperature and humidity gradients down the upper airway are increased, so heat and moisture exchange remains efficient.[2] The limits of the heat- and moisture-exchanging capacity of the upper airway have not been fully evaluated, and adaptation may occur with time. During exercise (because of an increased minute volume), additional demands are put on the heat- and moisture-exchanging function, and bronchospasm or rhinorrhoea may result.[5,6] Exercise-induced bronchospasm has been reduced by additional humidification in sensitive individuals.[7]

The Mucociliary Elevator

The bronchial tree is lined with mucus-secreting ciliated epithelium. Mucus is cleared in a cephalad direction by the cilia. A cilium is a hairlike structure, approximately 6 μ in length and with several short 'claws'on its tip. Normally the cilia are bathed in the mucus of the periciliary mucus layer, which is of low viscosity. The cilial claws extend through this layer and are in contact with a high viscosity mucus layer. The cilia beat in a regular, coordinated manner to clear this mucus. Effective mucus clearance depends on ciliary integrity and beat, and on the depth and viscosity of the periciliary mucus and the quantity and viscosity of the mucus layer. All these factors are affected by the state of hydration of the system.[8]

Inadequate Humidification

Underhumidification of inspired gases is most obvious during artificial ventilation with dry medical compressed gases to which no additional humidification has been supplied. Under these circumstances, a number of changes occur as a result of heat loss, moisture loss, and altered pulmonary function.

Most of the heat lost from the respiratory tract occurs as a result of vaporisation of water. Heat lost in this way may cause a drop in body temperature,[9] particularly in vulnerable groups such as infants, young children,[10] and critically ill patients whose thermoregulatory mechanisms are already disturbed. Humidification of inspired gases reduces the drop in body temperature seen following surgery.[9,11] This has implications for postoperative oxygen consumption[12,13] and associated problems, such as profound vasoconstriction.[9,14]

Ventilation with dry gases leads to considerable loss of moisture from the respiratory tract; this loss may be great enough to reduce body weight due to dehydration.[15] Dehydration of the upper airway is associated with a number of histologic changes,[15,16] and these are detailed in Table 1. The most important functional result of these changes is impaired function of the mucociliary elevator.[15,17-22] This leads to sputum retention and atelectasis. Damage to the basement membrane and the cells of the airway leads to tissue disruption, bronchiolar collapse, and ultimately atelectasis.

Disturbances of both structure and function may occur after as little as 10 minutes of ventilation with dry gases.[18] The degree of damage is directly

Table 1. The Histologic Lesions Reported during Ventilation with Dry Inspired Gases

Destruction of cilia
Damage to mucus glands
Disorganisation and flattening of both columnar and cuboidal epithelium
Disruption of basement membrane
Degeneration of cellular cytoplasm and nucleus
Desquamation of cells
Mucosal ulceration
Reactive hyperaemia

proportional to the duration of ventilation with dry gases, and recovery time is inversely proportional to the duration of ventilation.[15] Superficial repair, such as repair of ciliary damage, may take 2-3 days, while repair of full thickness lesions takes 2-3 weeks.[16]

Ventilation with dry gases shifts the isothermic saturation boundary downwards.[23,24] This is associated with changes in pulmonary mechanics that lead to hypoxaemia. Functional residual capacity and static compliance fall and alveolar-arterial oxygen tension difference rises. These changes appear to be due to atelectasis and an increased intrapulmonary shunt.[22,24,25]

Surfactant activity is impaired during dry gas ventilation. This results in a rise in surface tension and further impairs gas exchange.[17,22] Dry gases also act as potent bronchoconstrictors in sensitive individuals.[15,17] In addition, there is some evidence that humidification of inspired gases may reduce the incidence of postoperative pulmonary complications; however, this is inconclusive.[9,26-29] The extent of all these changes is related to the inspired humidity and the duration of ventilation.[11,16]

Excessive Humidification

Excessive artificial humidification of inspired gases can disrupt the dynamic equilibrium of heat and moisture exchange within the upper airway and replace this with a more static environment.[3] Under these circumstances, heat and moisture may be added to the body, and changes in pulmonary function may occur. This has been reported most commonly with misused or defective humidifiers.

Heat may be added to the respiratory tract if the inspired gases are heated to above body temperature and saturated with water vapour. As in the case of inadequate humidification, this addition of heat is particularly important in neonates, children,[30,31] and patients with impaired thermoregulatory mechanisms. There have been reports of mucosal heating or burning[2,32] that has led to pulmonary oedema and airway stricture formation.[32] A rise in body temperature has also been reported.[2] Conversely, the administration of large quantities of water to the respiratory tract may cause a fall in body temperature if the water is at or below room temperature.

The excessive humidification of inspired gases not only reduces insensible water loss but may also cause water to be added to the body, leading to water overload. Water intoxication has been reported with aerosol humidification.[33,34] The use of aerosols of water at room temperature may cause clinically important mucosal cooling,[16] and condensation of water droplets within the airways may lead to atelectasis. Ciliary damage may also result from excessive humidification.[16] However, the most important effect of excessive humidification is to render the mucociliary elevator less efficient. This may result from the production of large quantities of mucus, which may exceed the capacity of the mucociliary elevator, or from excessive hydration of the periciliary mucus layer so that transportation of the more viscid mucus is impaired.[18,20,26,35]

The upward movement of the isothermic saturation boundary leads to changes in pulmonary function. These changes include falls in functional residual capacity and in static compliance, leading to atelectasis and arterial hypoxaemia.[23,36,37] Surfactant activity is decreased to a more marked extent than with inadequate humidification. This may be due to the inhibition of surfactant production by atelectasis or the inactivation or dilution of surfactant by excessive water.[16]

Tolerated Range of Humidification

An optimal level of humidification is difficult to determine. The literature on humidification requirements contains mostly work in animals.[17,19,20,23,38] The studies in man[22,39] suffer from problems with small numbers, lack of data on the patients involved, and inadequate measurement techniques. Little new information is available on upper airway function and its changes in disease states and on advances in measurement techniques. Both underhumidification and excessive humidification are associated with harmful effects. However, I believe that many of the problems associated with excessive humidification have been the result of misuse of hot water humidifiers or nebulisers. The improved design and more rational use of these humidifiers may have reduced the incidence of such problems.

The upper airway normally tolerates a wide range of atmospheric conditions. Factors that affect the efficiency of normal heat and moisture exchange are the temperature and relative humidity of inspired gas, the inspired tidal and minute volumes, and the state of the airway (for example, the presence of a tracheal tube). Extremes of any of these factors challenge the ability of the nose and upper airway to condition inspired gases adequately. The maintenance of normal levels of temperature and humidity may preserve mucociliary and pulmonary function in normal individuals. This, however, has not been verified, and the optimal ranges of temperature and humidity in patients with abnormal, diseased, or bypassed upper airways remain uninvestigated. In disease states, the range of conditions tolerated by the upper airway is likely to be narrowed. However, the effects of any change cannot be predicted with current knowledge of upper airway function.

The effects of the level of humidification on the function of the mucociliary elevator are central to the rational use of humidifiers. Measurement of mucociliary function should provide some assessment of the adequacy of heat and moisture exchange. Mucociliary function is complex, and its measurement equally complex; however, an indication of mucociliary elevator function can be gained by monitoring sputum volume and character with a sputum score such as that in Figure 1.[40] In this way, the adequacy of humidification can be measured and recorded in a more objective way, and any necessary changes in humidification technique can be made appropriately.

Abundance	0	+	++	+++	++++
Viscosity	0	+	++	+++	++++
Purulence	0	+	++	+++	++++
Blood	0	+	++	+++	++++

Fig. 1. A sputum score to monitor the adequacy of heat and moisture exchange. 0 = absent; + = small amount; ++++ = copious.

How To Humidify Inspired Gases

Devices that condition inspired gases have been categorized into three groups on the basis of their water output:[41] (1) those delivering approximately 10 g/m³ of water to reproduce environmental humidity, (2) those capable of delivering at least 30 g/m³ of water and intended for use when the upper respiratory tract is bypassed, and (3) those capable of delivering in excess of 44 g/m³ of water.

Many different types of humidifier are available. The properties desirable in a humidifier[5] are detailed in Table 2, and the ability of the different humidifiers to fulfill these requirements is summarised in Table 3.

Table 2. Properties of an Ideal Humidifier

The delivery of adequate heat and moisture
Safety—no risk of malfunction, electrical hazard, misconnection, disintegration, inhalation debris, leakage, or drug interaction
No microbiologic risk
Suitable physical properties—size, resistance, functional dead space, and internal compliance
Convenience—use, cleaning, and storage
Economical—capital and running costs

Table 3. A Comparison of Different Humidification Techniques

	Cold-Water Humidifier	Hot-Water Humidifier	Nebuliser	HMEF*
Moisture output (g/m^3)	15-20	35-50	20-1000	25-35
Body temperature maintenance	Poor	Very good	Poor	Good
Safety	Good	Electrical overheating	Electrical	Connection
Microbiologic risk	Reservoir	Reservoir circuit	Reservoir circuit	Low
Internal compliance	Low	High	Moderate	Low
Convenience	Fair	Poor	Poor	Good
Cost	Moderate	High	High	Low

*HMEF = heat- and moisture-exchanging filter.

Cold-Water Humidifiers

In a simple, cold-water humidifier, gas directed over the surface of water at room temperature takes up a modest amount of water vapour (Fig. 2). The efficiency of the device can be increased by bubbling the gas through water or by introducing a wick, both of which increase the surface area of water exposed to gas. The absolute humidity of the gas leaving the vaporiser is limited by the surface area of the gas-water interface and the water temperature (allowing for the cooling effect of vaporisation). Recently developed devices can achieve 100% relative humidity, but the gas is unheated; so, the absolute humidity of the gas leaving the device remains inadequate when the respiratory tract is bypassed. Some risk of microbiologic contamination of the water reservoir exists.[42]

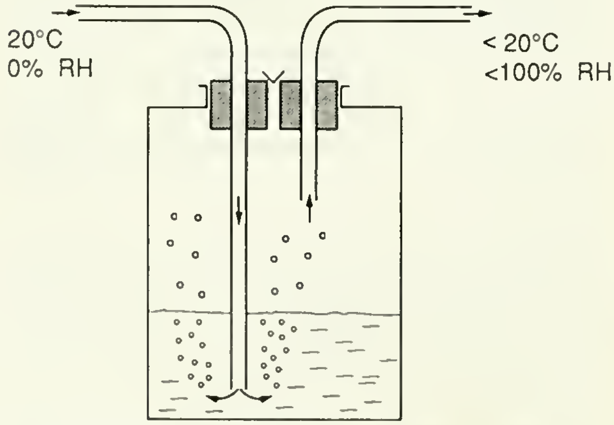


Fig. 2. A cold-water bubble humidifier of the type often used to humidify low-flow oxygen.

Hot-Water Humidifiers

Hot-water humidifiers produce gas saturated with water vapour at above room temperature.[43-45] Different heat sources are used in different devices. Some have submerged heat sources; others use heating plates (Fig. 3). Most devices are servo-controlled to maintain the water temperature at a preset value. This is measured at the catheter mount and allows for cooling along the tubes of the breathing circuit. The resulting condensate requires drainage and may provide a nidus for bacterial colonisation.[46] Hot-water humidifiers condition inspired gases for a variety of applications. Their disadvantages include high internal compliance and resistance to breathing;[47] the risk of thermal injury or electrical problems, bacterial colonisation;[48] and lack of convenience. In addition, heated humidifiers may degrade volatile anaesthetic agents, which limits their perioperative use.[49]

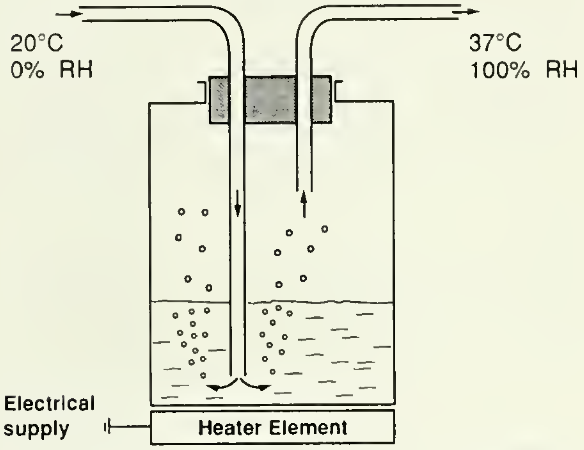


Fig. 3. Diagram of a heated bubble-type humidifier. Efficiency may vary.

Nebulisers

Nebulisers do not produce a water vapour but an aerosol of water droplets, which may be heated. This aerosol can be produced in three ways. Gas-driven nebulisers use the bernoulli effect to aspirate water from a reservoir into a gas jet;[41] a mechanical nebuliser uses a rotating disc to spin water radially onto pillars designed to break droplets up into a fine aerosol;[50] ultrasonic nebulisers[51] employ a submerged transducer to produce cavitation within the water reservoir, and produce an aerosol where the cavity breaks the water surface (Fig. 4). The administration of water droplets to the respiratory tract may result in excessive humidification or infection with microorganisms,[52] and the fate of these droplets within the respiratory tract is poorly understood. For these reasons, I believe that the use of nebulisers in the patient whose airway is bypassed can be hazardous.

Heat and Moisture Exchangers

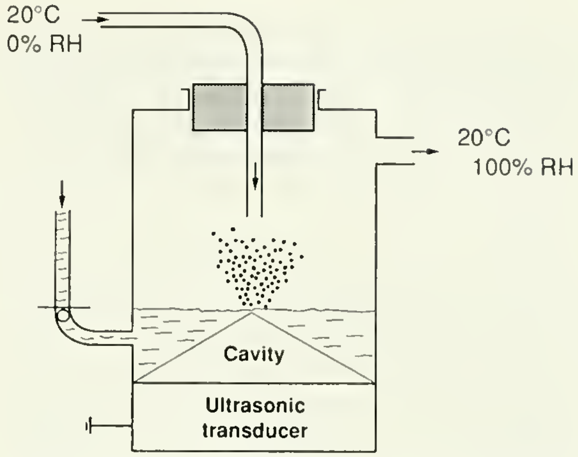
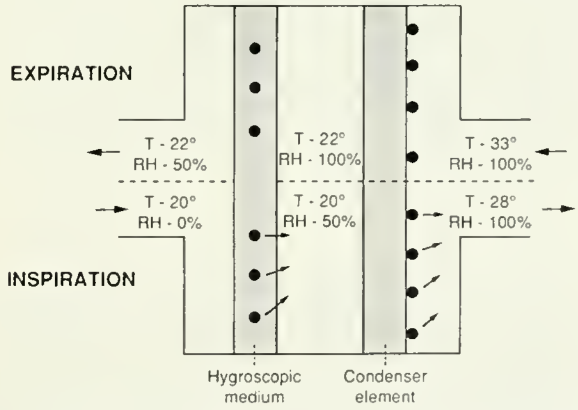


Fig. 4. Diagram of an ultrasonic nebuliser. The device (when functioning properly) saturates the gas stream and produces a dense stable aerosol.

Heat and moisture exchangers (HMEs) conserve heat and moisture during expiration and return these to the inspired gases in a way analogous to normal upper airway function.[53,54] It has been suggested that HMEs should retain at least 70% of expired moisture.[55,56] HMEs operate in one of three ways.[4] In a condenser humidifier, water vapour in expired gases condenses onto the relatively cool surface of the element; this water subsequently re-evaporates on inspiration. The element of a condenser humidifier has a high thermal conductivity; so, the gains and losses of latent heat are quickly compensated, and the temperature drop across the element is maintained.[57] Condenser humidifiers have been largely superseded by more efficient HMEs. Hygroscopic HMEs (Fig. 5) have elements composed of paper, foam, or some other substance with a relatively low thermal conductivity, that has been impregnated with a hygroscopic chemical (usually calcium chloride or lithium chloride). The



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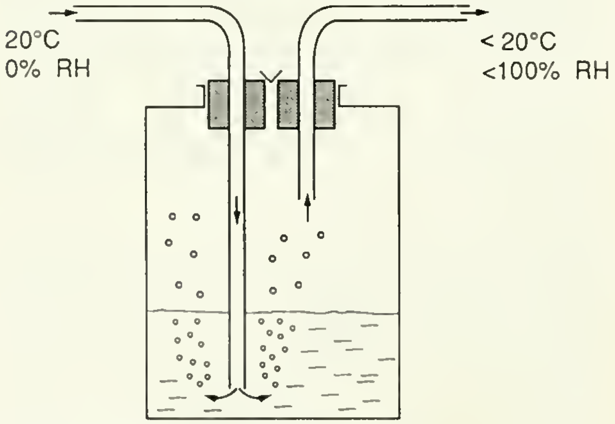

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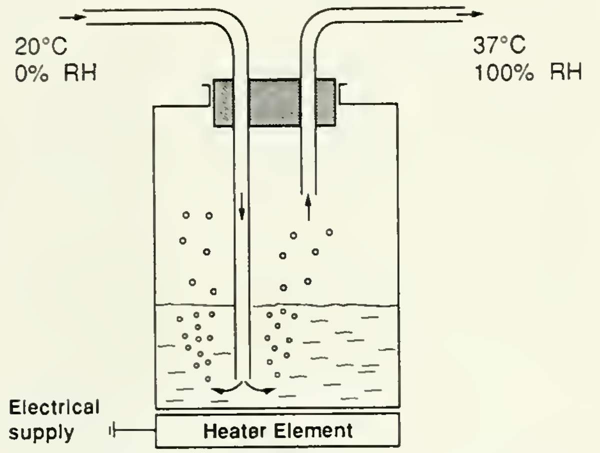

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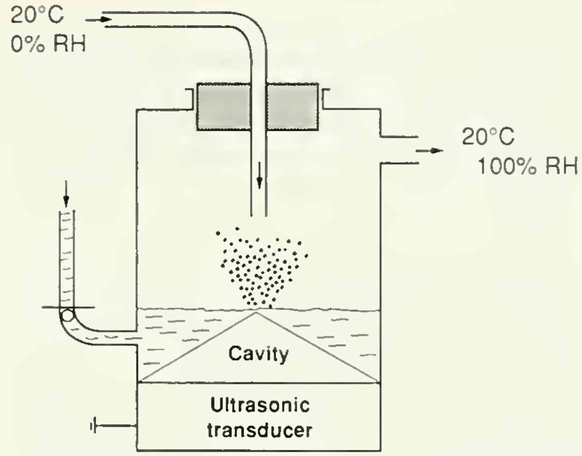

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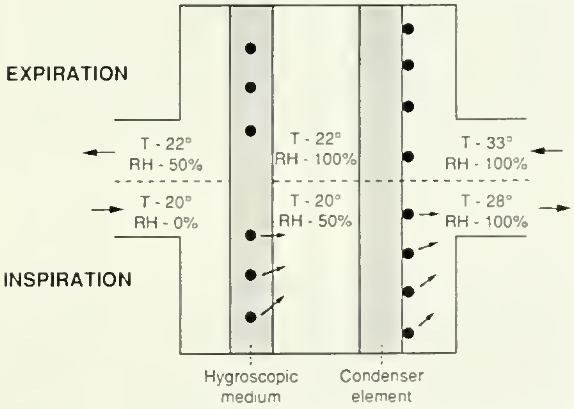

Fig. 5. Diagram of a hygroscopic heat and moisture exchanger.

element acts as a condenser humidifier, but efficiency is increased by the hygroscopic material, which adsorbs additional water on expiration. This water is then taken up by the inspired gases.[58] Hydrophobic HMEs (Fig. 6) have a water-repelling element with a large surface area and a low thermal conductivity. Because the latent heat of vaporisation is, thus, taken directly from the element and its surroundings when water is vapourised, a temperature gradient develops within the element itself. Gas leaving the device is saturated at the lowest temperature achieved; so, hydrophobic HMEs are more efficient than simple condenser humidifiers.[59,60] True hydrophobic HMEs are also efficient microbiologic filters.[61]

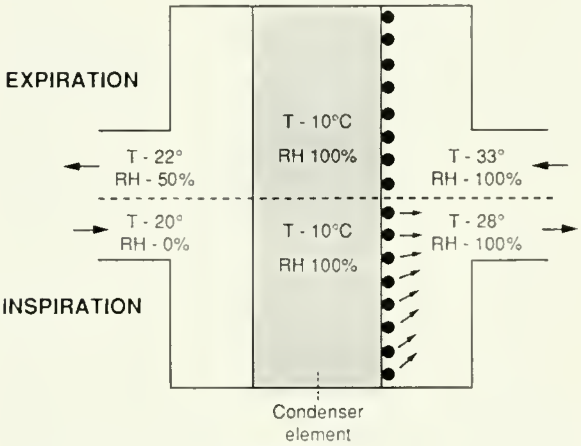

Fig. 6. Diagram of a hydrophobic heat and moisture exchanger.

Comparative investigations of heat and moisture exchangers have demonstrated that most are able to humidify inspired gases to a level similar to that of normal nose-breathing subjects at rest.[61-65] However, their efficiency depends on the tidal and minute volumes. Heat and moisture exchangers present a physical barrier to the passage of bacteria, but condenser humidifiers and hygroscopic HMEs are not microbiologic filters.

Applications

The clinical applications for which different types of humidification devices are suitable are summarised in Table 4.

Table 4. Suggested Applications for Different Types of Humidifier

Humidifier	Restrictions	Clinical Applications
Cold-water humidifiers	Intact airway O_2 therapy with abnormal airway or high fresh-gas flow	O_2 therapy
Hot-water humidifiers	Ventilatory support with abnormal airway, high V_{min}, large fresh-gas flow, large gas volumes	Oxygen tents Head boxes ICU* Anaesthesia Paediatrics CPAP
Nebulisers	High fresh-gas flow Large volumes Sputum clearance	Oxygen tents Head boxes CPAP HFV Physiotherapy
Heat and moisture exchangers	Ventilatory support with normal airway and normal minute ventilation	Infection control ICU Anaesthesia Transport

*ICU = intensive care unit; CPAP = continuous positive airway pressure; HFV = high frequency ventilation.

Cold-water humidifiers do not provide sufficient humidification to replace the patient's own airway and, consequently, are unsuitable for use in patients whose airways are bypassed by tracheal intubation. Cold-water humidifiers are most frequently used to humidify supplemental oxygen administered to spontaneously breathing patients. Although humidification may be unnecessary in this situation, it should be considered if the patient has abnormal airway function or the supplemental oxygen flow is high.

Hot-water humidifiers are more widely applicable, but the ability to vary the absolute humidity of these devices is not fully utilized. They are used in patients requiring controlled ventilation during anaesthesia or intensive care in whom the requirement for humidification may be high (such as in patients requiring high minute volumes and patients with abnormal airway function). Because of the large volume of water present in aerosols produced by nebulisers, they may be potentially hazardous. Nebulisers are, therefore, most frequently used to humidify the gas provided to large-volume tents and hoods, or gas at high flowrates such as during high frequency jet ventilation[66] and continuous positive airway pressure. Nebulisers may also be used to provide high levels of humidification for brief periods to patients with chronic lung disease to enhance sputum clearance during physiotherapy. HMEs are able to condition inspired gases. The addition of a filter, however, provides microbiologic protection, and heat- and moisture-exchanging filters (HMEFs) are preferable to simple HMEs. HMEFs are suitable for patients in whom humidification requirements are likely to be normal. This includes patients without significant chest or upper airway disease who do not require ventilation with high minute volumes (ie, ventilation in excess of 10 L/min, although this may vary with the device). Because HMEFs return essentially physiologic levels of humidification to the patient's airway, their use may be continued until the patient's humidification requirements change.

I do not believe that clinical comparisons of different humidification techniques have been performed on a rational basis. Recent comparisons of HMEs and hot-water humidifiers have had methodologic problems,[39,67] but have not demonstrated significant differences between the techniques.

Why Filter Inspired Gases?

Normal Mechanisms of Filtration

In addition to their heat and moisture exchanging function, the nose and upper airways act as an efficient filter. Particle filtration is accomplished by several mechanisms. Turbulent flow within the nose allows larger particles to impinge on the large surface area of mucous membrane. In the upper airway, mucus flow collects further debris that is cleared by the mucociliary elevator. Coughing also clears mucus from the upper airway.[8] Microbiologic filtration is achieved by the lymphoid tissue of the tonsils and adenoids and by protective mediators within the mucus itself.[5,6]

Bypassing the nose and upper airway, therefore, skirts the area responsible for filtration as well as the heat- and moisture-exchanging surfaces. This allows whatever particulate and microbiologic debris are to reach the lower airways. This may occur at a time when the patient is immunocompromised due to illness or surgery or is exposed to additional particulate or microbiologic hazard from the use of breathing circuits, ventilators, and artificial humidification devices. Realisation of this risk has led to the increased use of filters in respiratory care.

Filters

A filter for use in a breathing system should prevent passage of contaminated body fluids. It should, therefore, filter bacteria and viruses as well as particles and prevent passage of water. The attributes of an ideal breathing system filter have been described.[68] All filters have pores to reduce the resistance of the device. The size of these pores is important in preventing the passage of water.

Filters remove gas-bourne particles in three ways.[68] Large particles are removed by direct interception by the pores of the filter medium. Bacteria passing through the filter medium have a momentum that leads to impaction on the filter. Once impacted, the particles are held on the membrane by weak electrostatic forces. Very small particles travel by Brownian movement and thus behave as if they were larger particles. These particles are also intercepted by the filter and held by electrostatic forces.

Basically two types of filter material are used in breathing circuit filters.[68] Hydrophobic membranes have small pores, rely on naturally occurring electrostatic forces within the filter medium, and do not allow the passage of liquid water through the membrane. The alternative filter material is called an electret and is produced by inducing a permanent electrical polarity into a felt-like material. This polarity enhances removal of charged particles, but the pore size of an electret is larger than that in a hydrophobic filter. As a result, liquid water is able to pass through an electret, reducing filtration efficiency.

Applications

Filters are widely used with humidification devices in an effort to control contamination risks. Nosocomial infections are due to a variety of factors,[69] of which contamination of respiratory equipment is only one. The impact of filter use on the incidence of nosocomial pneumonia, therefore, varies

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Monitoring of Pressure, Flow, and Volume during Mechanical Ventilation

Martin J Tobin MD

I. Introduction
II. Pressure Monitoring
  A. Pressure-Sensing Devices
  B. Pressure-Time Recordings
III. Volume and Flow Monitoring
  A. Volume-Measuring Devices
  B. Flow-Sensing Devices
  C. Problems with Volume Measurements
  D. Volume-Based Indices
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V. Display of Monitored Data

Introduction

Accurate measurement of airway pressure, airflow, and volume provides information necessary for effective management of a mechanical ventilator. These variables also need to be carefully monitored to detect ventilator malfunction and to minimize the risk of ventilator-induced complications.[1]

Pressure Monitoring

Pressure-Sensing Devices

Airway pressure can be measured with gravity-dependent fluid manometers, aneroid manometers, or electromechanical pressure transducers.[2] Fluid manometers are rarely, if ever, employed in measurement of airway pressures in critically ill patients. Although aneroid manometers are still employed to obtain 'spot' measurements of airway pressure, such as maximum inspiratory pressure in a patient being considered for a weaning trial, electromechanical transducers are now widely employed in mechanical ventilators. Many of the modern, microprocessor-based ventilators (Bird 6400ST, Bear 5, Hamilton Veolar, Puritan-Bennett 7200) employ a solid-state integrated silicon-wafer pressure transducer manufactured by the Microswitch Division of Honeywell (Douglas DeVries of Bird Products, Warren Sanborn of Puritan-Bennett, David Thompson of Hamilton, Tom Westfall of

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Dr Tobin is Professor of Medicine, Loyola University of Chicago, Stritch School of Medicine, and Program Director, Division of Pulmonary and Critical Care Medicine, Edward Hines Veterans Affairs Hospital, Hines, Illinois.

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The author has no financial interest in any of the products mentioned.

Bear Medical, personal communications, 1991). This transducer is stated to have an accuracy of ± (0.1 cm H_2O + 3% of reading), while a target pressure can be achieved with an accuracy of ± (2 cm H_2O + 3% of reading) (Warren Sanborn, Puritan-Bennett, personal communication, 1991). For example, if trigger sensitivity is set at −2 cm H_2O, the actual range is 1.84-2.16 cm H_2O. Likewise, if the target for pressure support ventilation is set at 20 cm H_2O, the actual range is 17.4-22.6 cm H_2O.

Pressure-Time Recordings

Monitoring of airway pressure is particularly valuable in patients receiving mechanical ventilation.

Airway Pressure Profile—By inspecting the airway pressure tracing, one can determine the ventilatory mode being employed. With spontaneous breathing, airway pressure decreases during inspiration and increases during expiration; however, these fluctuations should be very small unless the ventilator circuit poses a marked resistance. During controlled mechanical ventilation, increases in airway pressure occur at regular intervals without a preceding negative pressure deflection. During assisted ventilation, a negative pressure deflection (reflecting the patient's effort) occurs immediately before the rise in airway pressure; if the patient's respiratory efforts are less frequent than the back-up rate of the ventilator, some of the breaths show pressure profiles of the form seen with controlled mechanical ventilation. During synchronized intermittent mandatory ventilation, a mixture of airway pressure profiles is seen—some of the spontaneous breathing variety and others of the type observed with ventilator assistance. A plateau in airway pressure is expected during both pressure-support ventilation and pressure-control ventilation, but inspiratory time is constant with the latter whereas it is of variable duration with the former.

A continuous recording of airway pressure can provide valuable information regarding the amount of active respiratory work being performed by a patient receiving ventilator assistance. During controlled ventilation in a completely relaxed patient, the ventilator develops all of the pressure necessary to overcome the resistive, elastic, and inertial properties of the respiratory system. In this situation, the airway pressure tracing shows a smooth rise, remains convex upward, and is highly reproducible from breath to breath (Fig. 1[3]). During assisted ventilation, the patient usually triggers the ventilator by generating isometric pressure of −1 to −2 cm H_2O. However, the inspiratory muscles do not stop contracting once the ventilator has been triggered; instead, they continue to contract throughout a large portion of the mechanically delivered breath.[4] The degree of deformation and scooping of the airway pressure tracing provides a means of monitoring the amount of effort expended by a patient during the period of ventilator support. In particular, excessive scalloping of the airway pressure tracing suggests an inadequate inspiratory flow setting. On the other hand, a very fast rise in airway pressure suggests an inappropriately high flow setting.

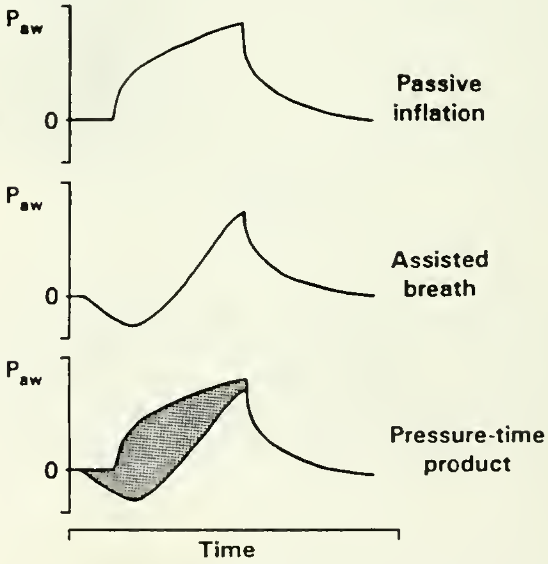

Fig. 1. Airway pressure (P_{aw}) tracings during controlled mechanical ventilation in a completely relaxed patient (top) and during an assisted breath (middle). The shaded area in the bottom tracing is the pressure-time product of the inspiratory muscles calculated as the difference in area subtended by P_{aw}-time curve in the presence (middle) and absence (top) of the inspiratory muscle activity. (Reproduced from Reference 3, with permission.)

If one assumes that the energy required to inflate the respiratory system is similar under passive and active conditions, energy expenditure of the patient's respiratory muscles can be estimated by subtracting the area under an inflation pressure-time curve generated when the patient is contributing to the work of inspiration from the area under a curve generated in the absence of inspiratory muscle activity (Fig. 1). This method of estimating energy expenditure is valid only if the active and passive curves are generated under conditions of identical inspiratory flow, tidal volume, and frequency, and provided that the mechanical properties of the lungs and chest wall remain unchanged between the generation of the two curves.

Peak Airway Pressure—In a ventilator-supported patient, alterations in peak pressure at the airway opening are sensitive indicators of abnormal pulmonary mechanics (provided ventilator-delivered tidal volume and inspiratory flow are unchanged) because peak pressure is influenced by a number of factors (Table 1). If an elevated peak airway pressure is detected, additional maneuvers such as airway occlusion should be performed to elucidate the etiology.[5] A decrease in peak airway pressure may result from a cuff leak or vigorous spontaneous efforts by the patient.

Table 1. Functional Disturbances Causing an Increase in Pressure at the Airway Opening

Functional Disturbance	Pressure Change
Increase in end-expiratory P_{st}* of respiratory system	Increased P_{st}
Increase in viscoelastic pressure gradient dissipations and/or time-constant inequalities	Increased difference between P_1 and P_{st}
Increase in airway resistance	Increased difference between peak P_{tr} and P_1
Increase in endotracheal tube resistance	Increased difference between peak P_{ao} and peak P_{tr}

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* P_{st} = static recoil pressure; P_1 = airway pressure immediately after brief (0.1-s) end-inspiratory pause; P_{tr} = tracheal pressure (measured distal to carinal end of endotracheal tube); P_{ao} = pressure at airway opening.

Peak airway pressure is used in the calculation of "effective dynamic compliance (C_{dyn})," which is derived by dividing ventilator-delivered volume by the peak airway pressure (minus PEEP).[1] This index is not a measure of true thoracic compliance because peak airway pressure also includes the resistive pressure component of the applied pressure.

However, C_{dyn} does reflect the effective inspiratory impedance of the respiratory system and is decreased by disorders of the airways, lung parenchyma, and chest wall. A more correct measurement of dynamic compliance is obtained by dividing tidal volume by the airway pressure immediately following end-inspiratory occlusion ($P_{occlusion}$); as before, PEEP must be subtracted from $P_{occlusion}$ when calculating compliance. $P_{occlusion}$ is the airway pressure that immediately follows end-inspiratory occlusion and is followed by a slow decline to plateau pressure.[6]

End-Inspiratory Occlusion Pressure—Measurement of the pressure at the airway opening during an end-inspiratory pause is routinely used in the calculation of "static" compliance of the total respiratory system.[7] To measure distending transthoracic pressure, the patient must be relaxed and making no respiratory effort. Conditions of zero gas flow are achieved by employing an inspiratory hold or by occluding the expiratory port long enough to allow the airway pressure to reach a constant value (usually 1-2 seconds). This pressure, commonly termed plateau pressure, represents the static elastic recoil pressure of the total respiratory system at end-inflation volume (P_{st}). If the patient is receiving external PEEP or demonstrates the presence of auto-PEEP, the PEEP needs to be subtracted from the plateau pressure in order to calculate the correct distending pressure. Volume measurements should be made at the connection port of the endotracheal tube, or the calculations need to take into account the volume stored in the distensible ventilator tubing (ie, the compression volume). Thus, total thoracic compliance is calculated as

$$\text{Compliance} = \frac{\text{Volume delivered} - (P_{plateau} - \text{PEEP}) \cdot \text{CV}}{P_{plateau} - \text{PEEP}},$$

where $P_{plateau}$ = plateau pressure, and CV = the correction factor for volume compressed in the tubing.

Examination of the plateau pressure during pressure support or pressure control ventilation also provides helpful information on ventilator performance. The failure to reach a plateau pressure should arouse suspicion of a leak, although such failure could also be due to inadequate flow.

End-Expiratory Occlusion Pressure—Occlusion of the airway at end-expiration provides important information because the pressure in the lungs and ventilator circuit equilibrate and the static recoil pressure of the respiratory system is displayed on the ventilator manometer. This pressure is normally zero, and a positive value indicates the presence of auto-PEEP.[8,9] This pressure is also called occult PEEP because it is not normally detected by the ventilator manometer, which is open to atmosphere. The major problem with the end-expiratory occlusion technique of measuring auto-PEEP is the difficulty in timing the occlusion, which needs to be performed immediately before the patient commences the next inspiratory effort. Consequently, it is most easily achieved in paralyzed patients receiving controlled ventilation. The Siemens 900C Servo ventilator has a special end-expiratory hold button to help with this measurement.[10]

Mean Airway Pressure ($\bar{P}_{aw}$)—Monitoring of hemodynamic status has progressed from measurements of systolic and diastolic pressure to measurement of mean pressure. Vasoactive therapy is increasingly being guided by the mean pressure response. Likewise, interest is growing in the use of $\bar{P}_{aw}$ to guide alterations in ventilator settings.[11-14] Changes in $\bar{P}_{aw}$ play a major role in mediating ventilator-induced alterations in gas exchange, cardiovascular function, and barotrauma. The importance of $\bar{P}_{aw}$ as a determinant of oxygenation was first emphasized by Boros et al,[11,12] who studied ventilator-supported neonatal infants and premature lambs with severe lung disease. They varied the inspiratory-expiratory ratio (I:E) and PEEP and found that improvements in oxygenation were directly related to increases in $\bar{P}_{aw}$ but not to I:E or PEEP per se. The improvement in oxygenation seen with increases in $\bar{P}_{aw}$ is thought to be due to recruitment of collapsed alveoli and/or the redistribution of lung fluid.

$\bar{P}_{aw}$ should be measured close to the endotracheal tube using a pressure tap directed perpendicular to axial flow. It can be calculated as the area under the pressure-time curve during both inspiration (t_i) and expiration (t_e) divided by the time of a total respiratory cycle (t_{tot}). It is considered a clinically useful composite of all of the pressures transmitted to the airway by the ventilator because it is affected by peak airway pressure (P_{peak}), duration of positive pressure ventilation, PEEP (auto- and external), inspiratory flowrate, pressure waveform, and compliance and resistance of the respiratory system. These factors can be reduced to three primary variables: inspiratory airway pressure (P_{awi}), fractional inspiratory time (t_i/t_{tot}), and PEEP. In a patient receiving passive mechanical ventilation with a constant flow, mean airway pressure is the following:

$$\bar{P}_{aw} = (\bar{P}_{awi})\,(t_i/t_{tot}) + \text{PEEP}\,(t_e/t_{tot}).$$

Several options exist to increase $\bar{P}_{aw}$ in a ventilator-supported patient (Table 2), but such measures are appropriate only if alveolar recruitment is still possible.[15] Although Boros et al[11,12] observed a direct relationship between mean airway pressure and improvement in oxygenation, all methods of

Table 2. Methods of Increasing Mean Airway Pressure ($\bar{P}_{aw}$)

Method	Cause of Increase in $\bar{P}_{aw}$
Increase in tidal volume	Increase in tidal elastic pressure
Increase in respiratory frequency	Reduction in t_e* produces dynamic hyperinflation and auto-PEEP
Decrease in inspiratory flowrate	Increase in t_i and decrease in t_e leads to dynamic hyperinflation
Addition of end-inspiratory pause	Increase in t_i and decrease in t_e leads to dynamic hyperinflation
Selection of decelerating flow profile	Greater proportion of average flow delivered earlier in inspiration
Addition of PEEP	Increase in airway pressure during expiration

*t_e= expiratory time; t_i = inspiratory time.

increasing $\bar{P}_{aw}$ may not be equivalent, and several lines of evidence suggest that maintenance of a certain level of PEEP may be an important co-factor.[15]

Volume and Flow Monitoring

Volume-Measuring Devices

Spirometers are commonly employed in pulmonary function laboratories to measure inspired and expired lung volumes.[16] These devices contain an expandable collecting chamber that alters dimension as gas enters or leaves it. One of the earliest models was the water-sealed spirometer, which consists of a bell suspended by a chain and pulley mechanism and sealed from the atmosphere by water. Movement of the bell with respiration is recorded on a kymograph. Dry rolling-seal spirometers are more commonly employed today.[17] These operate through displacement of a piston by gas entering and leaving the spirometer. Movement of the piston can be interfaced with a potentiometer that generates a change in voltage, which is proportional to change in volume.

In general, water-sealed or dry rolling-seal spirometers are not practical for monitoring ventilation in patients receiving mechanical ventilation. Their large bulk makes them inconvenient, and continuous recordings are difficult to obtain. However, a variant consisting of a bellows housed in a graduated, transparent plastic bell was employed in earlier Bennett ventilator models such as the MA-1. Accuracy of the latter spirometer was ± 100 mL, which is considerably less than the ± 20-mL accuracy of spirometers employed in pulmonary function laboratories.[2]

Small, hand-held spirometers are commonly employed to obtain spot checks of tidal volume (V_T) and minute ventilation ($\dot{V}_E$). These devices operate through a rotating vane mechanism that is driven by gas flow. Because they can be placed between the ventilator circuit and the patient's airway, they avoid the problem of inaccurate measurements resulting from compressed volume, which I discuss later. They are reasonably accurate at flowrates of 3-200 L/min, but flowrates above 300 L/min can damage the vanes and produce inaccurate results.[2,18,19] Inaccurate results may also result from accumulation of moisture; thus, the device should be dried between use by gently blowing dry gas through the apparatus. Popular examples of handheld spirometers include the Wright, Boehringer, and Ohmeda Respirometers.

Changes in thoracic volume can also be measured indirectly by monitoring rib cage-abdominal motion.[20] Most experience has been obtained with the respiratory inductive plethysmograph (NIMS, Miami Beach FL), which consists of two transducers placed around the rib cage and abdomen. These transducers generate frequency-modulated signals that are proportional to alterations in the enclosed cross-sectional area. The signals are sent to a demodulator/calibrator unit that converts them into a proportional voltage, which can be amplified and recorded. With careful calibration, measurements of tidal volume with the respiratory inductive plethysmograph are within 10% of spirometric measurements.[21,22]

Flow-Sensing Devices

Accurate measurement of airflow is vital to the effective operation of a mechanical ventilator and in patient monitoring. Flow is usually measured by a pneumotachograph (sometimes termed an anemometer) that can be attached to a mouthpiece.[23-26] To avoid the risk of impeding airflow, the resistance of a pneumotachograph should be < 1.5 cm $H_2O \cdot s \cdot L^{-1}$.[27] Likewise, dead space should be kept to a minimum (< 15 mL). A number of different flow-sensing devices are available.

Fleisch Pneumotachograph—The Fleisch pneumotachograph consists of a bundle of capillary tubes with parallel sides that provide a small fixed resistance to airflow.[23,24] Pressure taps, located at both ends of the capillary tubes, measure the pressure drop that develops as gas flows through the device (Fig. 2). Under laminar conditions, the relationship between the fall in pressure and flowrate is described by Poiseuille's law:

$$\dot{V} = \frac{\Delta P \pi r^4}{8nl},$$

where $\dot{V}$ is gas flow, ΔP is drop in pressure, r is radius of the resistor, n is gas viscosity, and l is the length of the resistor.

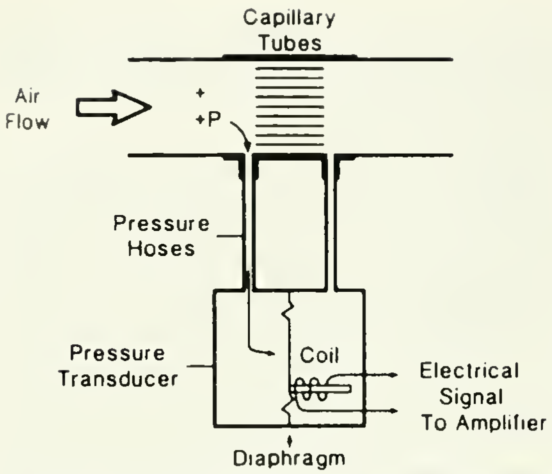

Fig. 2. Schematic representation of a Fleisch pneumotachograph with a differential pressure transducer. (Reproduced from Reference 24, with permission.)

Thus, if the configuration of the pneumotachograph and gas viscosity remain unchanged, the fall in pressure is directly proportional to flowrate. Because gases have different viscosities, calibration should be performed with the same mixture of gases that is to be used during measurement. Air and oxygen have viscosities of 184 and 200 poise, respectively, with the result that a pneumotachograph calibrated with room air yields a falsely elevated volume of 12-30% when measurements are made with the subject breathing 100% O_2.[24,28] A major problem with the Fleisch pneumotachograph is the accumulation of water and secretions in the capillary tubing that leads to alterations in resistance and inaccurate results. Warming the pneumotachograph to body temperature decreases condensation, but moisture accumulates over time and, thus, these instruments cannot be used for prolonged monitoring.

Screen Pneumotachograph—These devices perform in a manner similar to that of the Fleisch pneumotachograph but contain a fine-mesh screen rather than capillary tubes to produce a linear flow-pressure relationship. Reported accuracy is within ± 5% over a wide range of flows.[2] Purported advantages of screen pneumotachographs include decreased dead space, better frequency response, and ease of disassembly for cleaning purposes.[24] However, absorption of moisture produces clogging, and thus they need to be cleaned on a regular basis. This type of pneumotachograph is employed in the Siemens 900 series of ventilators (Siemens-Elema Ventilator Systems, Schaumburg IL). In one of the few studies examining the accuracy of ventilator-incorporated flow sensors, Synnott and Wren[28] found the output of the Siemens Servo 900C flow transducer to be virtually identical to that of a Fleisch pneumotachograph; unfortunately, they did not report the data on which they based this conclusion.

Orifice Pneumotachograph—Orifice pneumotachographs were developed because the screen-type and Fleisch pneumotachographs become clogged with water droplets and moisture. These sensors are small enough to create a measurable resistance to airflow, while being sufficiently large to permit the passage of water droplets. Two major forms of orifice pneumotachographs exist. One has a relatively large orifice that results in turbulent flow and thus needs to be used in conjunction with a linearizing electronic circuit. The second type of sensor incorporates an elastic flap in the middle of the orifice that produces mechanical linearization of flow, and, thus, is termed a variable orifice pneumotachograph (Fig. 3).[29,30] The variable-orifice pneumotachograph is employed in the Bird 8400ST ventilator (Bird Products Corp, Palm Springs CA) and in the Bicore Monitoring System (Bicore Monitoring Systems, Irvine CA).

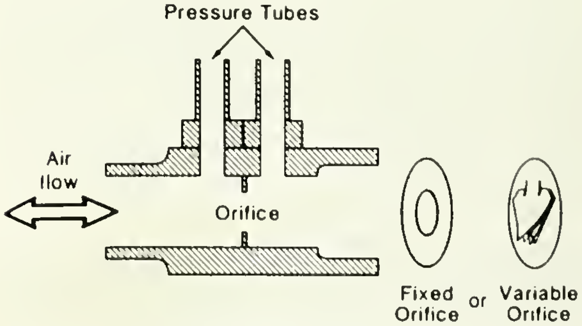

Fig. 3. Schematic representation of an orifice pneumotachograph. (Reproduced from Reference 24, with permission.)

Hot-Wire Pneumotachograph—This type of sensor consists of a thin wire, such as platinum, positioned in the middle of a tube and heated (Fig. 4).[24,31,32] As gases pass through the tube, the wire rapidly loses heat. Cooling is offset by an electronic circuit that adds more current to maintain a constant temperature in the wire. The amount of current added is proportional to the airflow. Purported advantages of this device are its lack of moving parts (which eliminates wear problems), the virtual absence of resistance to airflow, its extremely fast response time, and immunity to changes in gas density. However, accuracy is affected by altitude, ambient temperature, moisture, and turbulence. This type of sensor is employed in the Puritan-Bennett 7200 series of ventilators (Puritan-Bennett Corp, Overland Park KS), but little or nothing has been published to verify its accuracy.

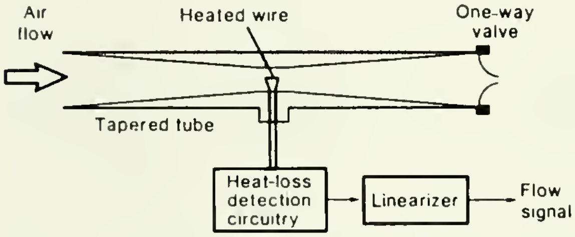

Fig. 4. Schematic representation of a hot-wire pneumotachograph. (Reproduced from Reference 24, with permission.)

Vortex-Shedding Pneumotachograph—The passage of air through a narrow tube containing an obstruction produces turbulent gas flow and creates waves or vortices. In this instrument, struts are strategically placed in a tube to create vortices that are detected by an ultrasonic beam placed downstream of the strut (Fig. 5). The degree of turbulence is proportional to the flowrate. In a detailed evaluation, Westenskow and Tucker[33] found the accuracy of a vortex flow sensor to be better than ± 2.5% over a flow range of 5-250 L/min. Variations in humidity, pressure, temperature, and gas composition caused the flowmeter to change by less than ± 4% of the reading.[33] Accuracy may deteriorate at high flowrates because the latter may create turbulence sufficient to produce confluent vortices. Inaccurate results may also result from condensation of water on the struts or sensors. This is the type of sensor employed in the Bourns hand-held ventilation monitor (Model LS-75) and in the Bear series of ventilators (Bear Medical Systems Inc, Riverside CA).

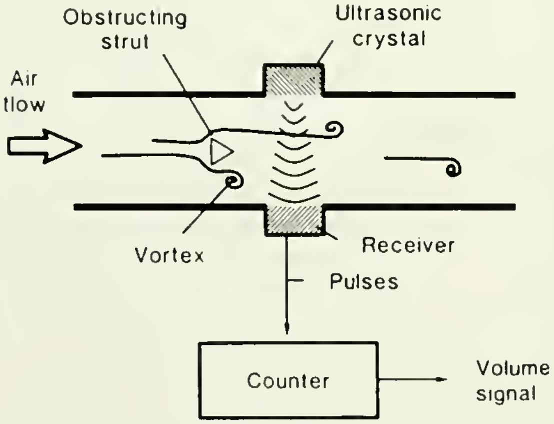

Fig. 5. Schematic representation of a vortex pneumotachograph. (Reproduced from Reference 24, with permission.)

Turbine Pneumotachograph—This device incorporates a turbine that rotates with gas flow.[32,34,35] As the turbine rotates, one of its blades interrupts a beam of light shining on a photocell, which in turn is translated into a measure of airflow. Advantages of this sensor are its reported immunity to turbulence, gas composition, water vapor, and temperature. A major limitation is the inertia of the blades in the turbine, which results in a poor response to rapid changes in airflow. Its signal has been shown to lag behind a Fleisch signal at the start of inspiration or expiration ("lag-before-start" effect) and to indicate flow for a few moments after the Fleisch signal has returned to zero at the end of inspiration or expiration ("spin-after-stop" effect)[34] (Alpha Technologies, Laguna Hills CA).

Problems with Volume Measurements

Compression Volume—The phenomenon of ventilator-circuit compression volume is an important consideration, especially in patients with marked abnormalities in lung mechanics. During the inflation phase of mechanical ventilation, pressure rises within the ventilator circuit, causing elongation and distention of the tubing and compression of the gas within the circuit. The volume stored in the circuit never reaches the patient, and, thus, the volume received is less than the set value. During the expiratory phase, however, the gas compressed in the circuit is released through the exhalation valve and is measured with the exhaled gases from the patient. As a result, recorded exhaled tidal volume is higher than that received by the patient. The magnitude of compression volume is determined by the internal volume of the ventilator, the volume of the in-line humidifier (and the amount of water in it), and the characteristics of the circuit tubing such as length, diameter and compliance, and inflation pressure.

Compression volume can be partitioned from the volume delivered to a test lung using a special valve system.[36,37] A compression factor or ratio can be calculated as

$$\frac{\text{compression volume}}{\text{inflation pressure}}.$$

In making this calculation, plateau rather than peak airway pressure is preferred, although this probably makes very little difference.[36,38] The compression factor is greater at high inflation pressures, such as occur with increased airway resistance or low lung compliance. For example, with the Marquest circuit the compression factor is 1.4 mL/cm H_2O at an inflation pressure of 19 cm H_2O compared with 2.1 mL/cm H_2O at a pressure of 63 cm H_2O. In addition, the compression factor is greater with disposable circuits. For example, at an inflation pressure of 63 cm H_2O, the Intertech, Marquest, and Inspiron disposable circuits have compression factors of 2.6, 2.1, and 1.9 mL/cm H_2O, respectively, compared with 1.4 mL/cm H_2O for the nondisposable Bennett circuit.[36,37] This difference is less marked at low inflation pressures.

The failure of the caregiver to allow for wasted compression volume may result in hypoventilation in ventilator-supported patients, particularly those with increased airway resistance or low compliance. It can also result in a falsely high value for calculated thoracic compliance. In addition, it may affect the calculations of O_2 consumption, CO_2 production, and dead space because it falsely elevates exhaled O_2 concentration and falsely decreases exhaled CO_2 concentration. Accordingly, a special valve system to separate exhaled gas from gas compressed in the circuit should be employed when making such measurements.

Some ventilators, such as the Bennett 7200 series, attempt to correct for compressed volume.[39] For example, for a set volume of 1,000 mL, compression factor of 4 mL/cm H_2O, and inflation pressure of 50 cm H_2O, the volume received by the patient is calculated as

$$\text{received volume} = 1{,}000 - (4 \times 50) = 800 \text{ mL}. \quad [1]$$

The ventilator can detect the 200 mL that remains in the circuit and adjust for this by automatically increasing the set volume of the next breath to 1,200 mL, which will result in an inflation pressure of 60 cm H_2O. Consequently, for the second and successive breaths,

$$\text{received volume} = 1{,}200 - (4 \times 60) = 960 \text{ mL}. \quad [2]$$

$$\text{received volume} = 1{,}240 - (4 \times 65) = 980 \text{ mL}. \quad [3]$$

$$\text{received volume} = 1{,}260 - (4 \times 67) = 992 \text{ mL}. \quad [4]$$

$$\text{received volume} = 1{,}268 - (4 \times 68) = 996 \text{ mL}. \quad [5]$$

$$\text{received volume} = 1{,}272 - (4 \times 68) = 1{,}000 \text{ mL}. \quad [6]$$

It can be seen that received volume should rapidly reach the set volume when this iterative approach is used.[39]

BTPS Correction—When gas exits a patient it has a temperature of 37°C, and it continually cools as it travels to the exhalation valve, with the result that gas temperatures vary from body temperature to ambient temperature in the ventilator system.[40] This becomes important because, according to Charles's law, gas volume varies directly with changes in absolute temperature. Gas volume measured at ambient temperature and pressure is said to be at ATPD or ATPS, depending on whether it is dry or saturated with water vapor.[41] Gas in the lung is at body temperature and pressure and saturated with water vapor (BTPS). Although the mass of gas does not change, the measured volume depends on the temperature at the point of measurement. If a difference in temperature exists between a flow- or volume-measuring device and the patient, a correction factor is necessary to report volume under BTPS conditions. For example, at an ambient temperature of 20°C, the factor to convert from ATPS to BTPS is 1.102.[41] If inspired gas is heated to body temperature, a correction factor is not needed. Likewise, if a pneumotachograph is placed close to the patient's airway, the exhaled gas is at about 37°C and saturated (100% humidity), and a correction factor is not needed.[24]

Because temperature and pressure sensors are incorporated into most ventilators, conversion from ATPS to BTPS should be an easy task. Ventilators differ considerably in attempts to correct for BTPS conditions. The expired volumes measured by the

Bear 5 ventilator can be corrected to ATPD or BTPS by employing a user-selectable switch. The flow sensor resides in a temperature-controlled chamber that heats and maintains gas at approximately 90°F. If BTPS is selected, the flow sensor signal will be corrected to 98.6°F (assuming a relative humidity of 99%). If ATPD is selected, the signal is not corrected, and a relative humidity of 15% is assumed (Tom Westfall, Bear Medical, personal communication, 1992). The Bird 8400ST ventilator corrects volumes to BTPD (Douglas DeVries, Bird Products Corp, personal communication, 1992). The conversion employed by the Puritan-Bennett 7200 ventilator is based on the assumption that the gas leaving the patient Y is 100% humidified at 37°C. Other ventilators, such as the Siemens 900C, do not include any BTPS correction factor (Bradford M Saunders, Siemens Life Support Systems, personal communication, 1992).

Humidity Effect—Measurement of gas volume is also affected by humidity. The measured volume of a given mass of gas is greater when the gas is saturated with water vapor than when it is dry. This effect of humidity is greater at higher temperatures, reflecting the increased ability of a warm gas to retain moisture. For example, if a given mass of gas measures 1,000 mL at 21°C under dry conditions, when saturated with water vapor it will increase to 1,007 mL at 21°C and to 1,022 mL at 37°C. Despite the importance of this effect, manufacturers generally do not incorporate humidity sensors into ventilators.

Volume-Based Indices

Minute ventilation is about 6 L/min in resting healthy subjects.[42] Because arterial carbon dioxide tension (P_{aCO_2}) is determined by the relationship between alveolar ventilation and CO_2 production, a high minute ventilation accompanied by hypercapnia indicates the presence of increased dead-space ventilation and/or increased CO_2 production.[1] Conversely, hypercapnia associated with a low minute ventilation should arouse the suspicion of decreased respiratory drive, structural abnormality of the thoracic cage, or respiratory muscle dysfunction. While a minute ventilation ($\dot{V}_E$) of less than 10 L/min is commonly used as a predictor of weaning outcome,[43] one should not rely on this criterion because it is associated with a high rate of false-positive and false-negative results.[44]

The measurement of $\dot{V}_E$ should be partitioned into its respiratory-frequency and tidal-volume components. In healthy subjects, respiratory frequency is approximately 17 breaths/min, and tidal volume is approximately 0.40 L.[42] An increased frequency is often the earliest sign of impending respiratory disaster,[45-47] and the degree of increase is proportional to the severity of the underlying disease.[48]

Patients who fail a trial of weaning from mechanical ventilation commonly display an increased respiratory frequency (f) and a low tidal volume (V_T).[49] These two measurements can be combined to produce an index of rapid shallow breathing, viz, the frequency-tidal volume ratio, or f/V_T. In a recent prospective study of patients being weaned from mechanical ventilation, f/V_T was superior to conventional predictors of weaning outcome.[50] Of the patients who had an f/V_T value > 100 breaths/minute/L, 95% failed a weaning trial, whereas 80% of the patients with lower f/V_T values were successfully weaned. As a method of assessing pulmonary performance in critically ill patients, f/V_T has a number of attractive features: It is easy to measure; it is independent of patient effort and cooperation; it appears to be accurate in predicting the ability to sustain ventilation; and, fortuitously, it has a rounded-off threshold value (100) that is easy to remember. The precise pathophysiologic basis of an elevated f/V_T in patients who fail a weaning trial is not known, but it may be a stress response reflecting an imbalance between respiratory neuromuscular reserve and respiratory demands.

Comparison of inspired and expired V_T is helpful in arousing suspicion of a leak. If expired volumes are substantially less than inspired volumes, one should consider a leak around the cuff of the endotracheal tube or in the circuit, or across the chest wall (a bronchopleural fistula). Additional evidence of a leak in a ventilator-supported patient includes a decrease in peak airway pressure and a fall in peak expiratory flowrate.

Flow-Volume Curves

In the pulmonary function laboratory, measurements of forced expiration are the most commonly used physiologic test of lung function. They play a major role in determining the nature of lung disease, defining its severity and progression over time, and assessing response to therapy.[16,51] A forced expiratory maneuver can be examined in terms either of expired volume as a function of time or of expired flow as a function of lung volume (flow-volume curve). Four factors determine maximum expiratory flow: (1) voluntary effort, (2) elastic recoil pressure of the respiratory system (which is the driving pressure for resting expiration), (3) resistance of the airways (which determines the frictional pressure loss), and (4) compliance of the airway at the site of collapse (this last factor can be ignored because disorders of compliance of the major airways rarely occur in the absence of a decrease in lung recoil).

Maximum expiratory flow-volume curves are commonly analyzed in terms of the flow at a particular volume, such as 50% or 25% of vital capacity (ie, 50% or 25% above residual volume). However, analysis confined to such numerical indices ignores most of the data in the curve. Instead, simple inspection of a curve and employment of pattern recognition (similar to the situation with electrocardiography) can rapidly yield important information. At a glance, one can see the scalloping that is characteristic of severe airflow limitation (without reviewing the numerical values of the various derived indices) or flow-volume profiles that suggest upper airway obstruction.

Flow-volume curves may also provide useful information in ventilator-supported patients, although this has attracted relatively little scrutiny. In contrast to ambulatory patients, in mechanically ventilated patients forced expiratory tests are impractical. However, useful information can be derived from examination of flow-volume curves obtained during spontaneous breathing.[52] In a relaxed patient, elastic recoil of the total respiratory system drives exhalation; resistance to flow is the only opposing force. Normal subjects display a smooth decrease in expiratory flow throughout expiration, whereas patients with airflow limitation show a curvilinear pattern (convex to the volume axis) (Fig. 6).[52,53] Many of these patients have auto-PEEP, and consequently their end-expiratory lung volume is higher than their relaxation volume. As a result, expiratory flow stops abruptly before the next mechanical inflation, producing an expiratory flow curve with a characteristically truncated appearance.

Determining whether external PEEP is inducing alterations in the flow-volume curve is also helpful from a diagnostic viewpoint. Patients with COPD and airflow limitation commonly develop auto-PEEP as a result of critical closure of the airways—the physiology of which has been likened to

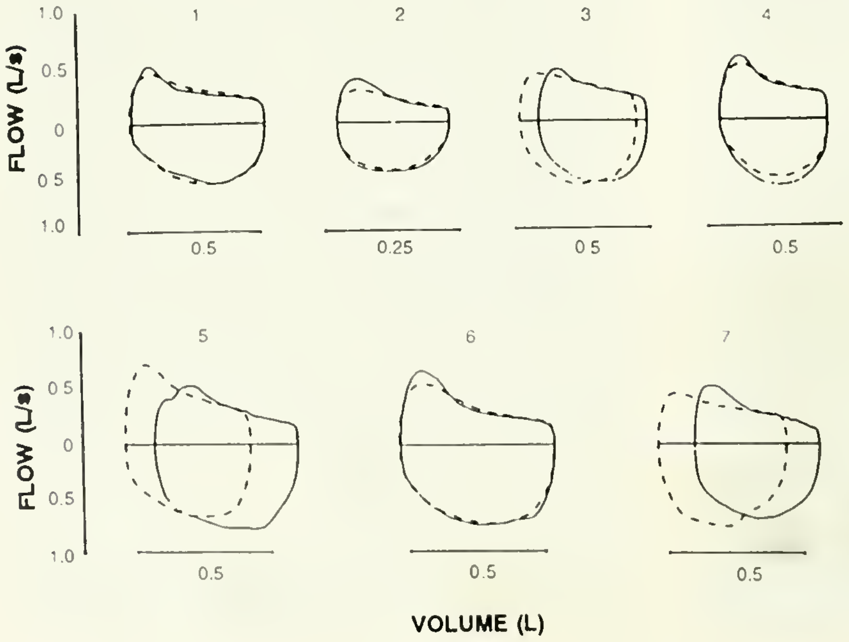

Fig. 6. Average tidal flow-volume loops in seven patients with COPD during control (solid line) and continuous positive airway pressure (CPAP) of 5 cm H_2O (dotted line). Note that expiratory flow-volume events were largely unaffected by CPAP in Patients 1, 2, 4, and 6, indicating the presence of flow limitation. (Reproduced from Reference 53, with permission.)

a waterfall, where the height of the waterfall reflects the height of the critical closing pressure.[54,55] In this situation, the driving pressure for flow throughout expiration is the level of auto-PEEP minus the critical closing pressure (height of the waterfall). Thus, the application of external PEEP at levels up to the level of auto-PEEP has no appreciable effect on expiratory flow, pressure upstream (alveolar pressure), end-expiratory lung volume, or the flow-volume curve (Fig. 6). In contrast, the effective expiratory driving pressure in patients without airflow limitation is elastic recoil pressure minus the pressure at the airway opening. Accordingly, the application of external PEEP will decrease the expiratory driving pressure and produce a decrease in expiratory flow at all lung volumes and an increase in end-expiratory lung volume (Fig. 7).

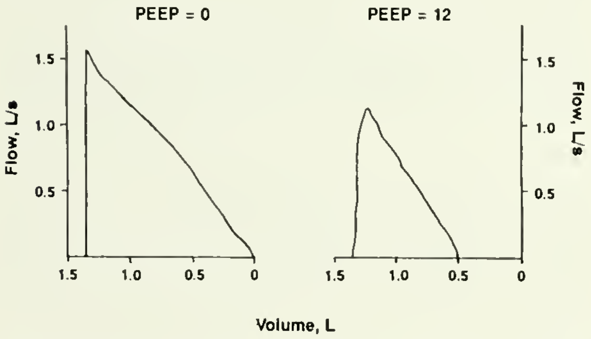

Fig. 7. Tidal expiratory flow-volume curves in a patient with adult respiratory distress syndrome receiving mechanical ventilation. Note the smooth decrease in flow throughout expiration (in contrast to Figure 6). The curve on the right shows the effect of adding 12 cm H_2O PEEP; note the decrease in expiratory flow and the increase in end-expiratory lung volume. (Modified from Reference 52, with permission.)

Bronchodilator therapy in ventilator-supported patients poses a problem not only in terms of delivery through an endotracheal tube, but also because universally accepted indices of bronchodilatation do not exist nor do we know what degree of change in an index reflects meaningful clinical improvement. In a recent study (1987), Gay et al[56] measured lung mechanics before and after the administration of aerosolized metaproterenol. They recorded airway pressure, flow, and lung volume during controlled inflation and stepwise deflations of the relaxed respiratory system at lung volumes between end-inspiration and static equilibrium volume. An increase in expiratory flow at a constant recoil pressure (ie, constant lung volume) was considered to represent bronchodilatation because the medication did not alter the pressure-volume relationships of the respiratory system. They arbitrarily selected a recoil pressure of 6 cm H_2O and noted the change in expiratory flow after drug administration. Expiratory flow increased by 59% (from 0.22 to 0.35 L/s), associated with a 25% decrease in auto-PEEP (from 9.6 to 7.2 cm H_2O) and a 0.2-L decrease in end-expiratory lung volume. Comparison of flow-volume curves before and after metaproterenol shows an increase in expiratory flow at comparable lung volumes and a decrease in end-expiratory lung volume (Fig. 8).[56]

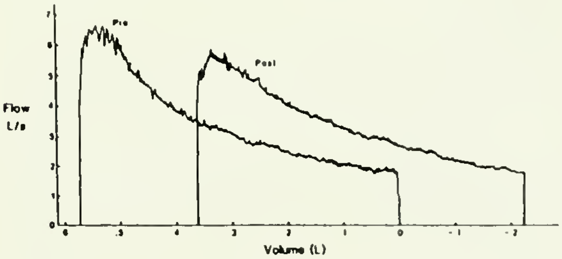

Fig. 8. Expiratory flow-volume curves in a patient with flow limitation. Note that bronchodilatation produces an increase in isovolume flow and a decrease in end-expiratory lung volume. (Reproduced from Reference 56, with permission.)

The flow-volume curve may also be helpful in indicating a need for endotracheal suctioning. We recently observed a sawtooth pattern on the flow-volume curve of an intubated patient with copious secretions that disappeared after suctioning. To determine the reproducibility of this observation, we undertook a preliminary study in 35 ventilator-dependent patients. Detection of a sawtooth pattern during spontaneous breathing strongly suggested the presence of secretions (positive predictive value, 94%), and the absence of this pattern suggested that secretions were unlikely to be present (negative predictive value, 77%).[57]

Flow-Time Recordings

A plot of flow versus time can provide information about both the ventilator and the patient

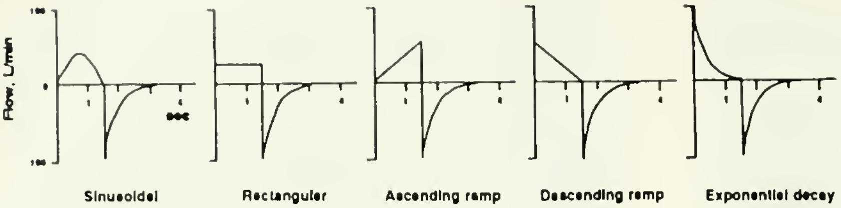

Fig. 9. Flow-time tracings during sinusoidal, constant (rectangular), ascending ramp, descending ramp, and exponential-decay flow waveforms. Horizontal axis is time in seconds. (Modified from Reference 38, with permission.)

(Fig. 9). If the patient is breathing spontaneously, a sinusoidal flow pattern is seen during inspiration.[38] Mechanical ventilation is usually delivered with a constant flow (retangular) pattern. Other profiles that can be produced include an ascending or descending ramp flow profile and an exponential-decay flow waveform during pressure-controlled ventilation. The relative advantages and disadvantages of different waveform patterns remain controversial.[58,59]

Inspection of the flow-time tracing may also indicate the presence of auto-PEEP. In the absence of auto-PEEP, a period of zero flow is evident before the next spontaneous or mechanical inhalation. When auto-PEEP is present, flow continues throughout expiration and is abruptly terminated by the onset of the next inflation (spontaneous or mechanical). However, a quantitative calculation of the level of auto-PEEP cannot be made from examination of the flow-time profile.

Location of Flow and Pressure Sensors

Flow and pressure sensors can be positioned at several locations within a ventilator delivery system: within the ventilator itself (on the inspiratory or expiratory circuit) or at the connection between the patient's airway and the breathing circuit. Several feet of narrow tubing may be used to connect a sensing port at the proximal airway with a transducer in the ventilator.

For monitoring purposes, sensors ideally should be located between the Y of the ventilator circuit and the endotracheal tube, as with the current location of flow sensors on the Hamilton Veolar and Amadeus ventilators (Table 3). More commonly, flow sensors are located on the expiratory limb of the ventilator. In this location, they cannot distinguish between the volume received by the patient and that compressed in the circuit, as I discussed earlier. The considerable discrepancy

Table 3. Type and Location of Flow and Pressure Sensors in Five Ventilators

	Flow-Volume Monitoring		Pressure Monitoring	
Ventilator	Sensor Type	Site	Sensor Type	Site
Bear 5	Vortex-shedding	Distal to exhalation valve	EMT*	Proximal airway
Bird 6400ST	Variable-orifice	At exhalation-valve outlet	EMT	Expiratory limb
Hamilton Veolar	Variable-orifice	Proximal airway	EMT	Inspiratory limb
P-B 7200a	Hot-wire	Distal to exhalation valve	EMT	Expiratory limb
Siemens 900C	Screen	Proximal to exhalation valve for $\dot{V}_E$ and expired V_T; inspiratory limb for inspired V_T	EMT	Inspiratory and expiratory limb

*EMT = electromechanical transducer; $\dot{V}_E$ = minute ventilation; V_T = tidal volume.

between volume displayed on a monitor and that received by a patient was demonstrated in two recent preliminary studies (abstracts). Figure 10 shows data by Gammage et al[60] of volume displayed on a ventilator monitor and that measured with a pneumotachograph placed between the Y and the airway connector. The ventilator was connected to a test lung with a low compliance (0.02 L/cm H_2O) and tidal volume was set at 1,000 mL. The discrepancy between displayed volume and the volume received varied among the ventilators, being greatest and most variable for the Siemens ventilator (23%). The smallest discrepancy occurred with the Hamilton ventilator (0.3%), reflecting the fact that its flow sensor is located between the Y and the airway connector. Data from a similar preliminary study by Branson et al[61] are shown in Figure 11, demonstrating the differences between set, displayed, and actual volumes for four ventilators attached to a test lung with a compliance of 0.01 L/cm H_2O. Again, the least discrepancy between displayed and measured volume was with the Hamilton ventilator. Although some of the ventilators employed in these two studies[60,61] attempt to compensate for compressed volume (such as the Puritan-Bennett 7200a and the Bear 5), the data in Figures 10 and 11 indicate that such compensation is imperfect. Another problem that arises when flow-volume sensors are placed at a distance from the patient's airway is the failure to appropriately correct for conversion from ATPS to BTPS conditions. Furthermore, location of flow sensors at a distance from the patient's airway increases the risk of inaccuracies due to leaks in the circuit. For the purpose of monitoring, manufacturers rarely if ever place flow sensors on the inspiratory side of a ventilator.

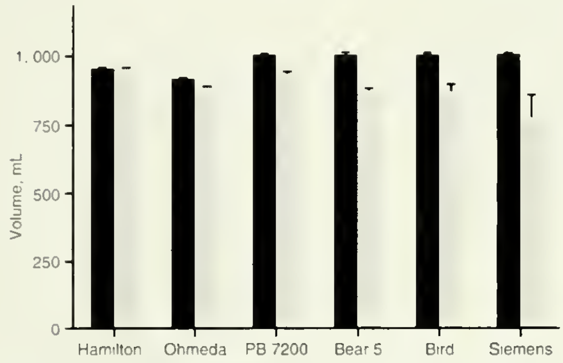

Fig. 10. Volumes displayed on a ventilator monitor (darkened bars) and that measured with a pneumotachograph placed between the patient Y and the airway connector (gray bars). Each ventilator was connected to a test lung with a low compliance (0.02 L/cm H_2O) and tidal volume was set at 1,000 mL. The ventilators are arranged in order of increasing discrepancy between displayed and measured volume. Values are mean + SD. (Based on data from Reference 60.)

Pressure can also be sensed at various locations. For monitoring purposes, pressure is sensed on the inspiratory side of the Hamilton Veolar ventilator, on the expiratory circuit of the Puritan-Bennett 7200a and Bird 6400ST ventilators, and at the proximal airway of the Bear 5 ventilator (the prox-

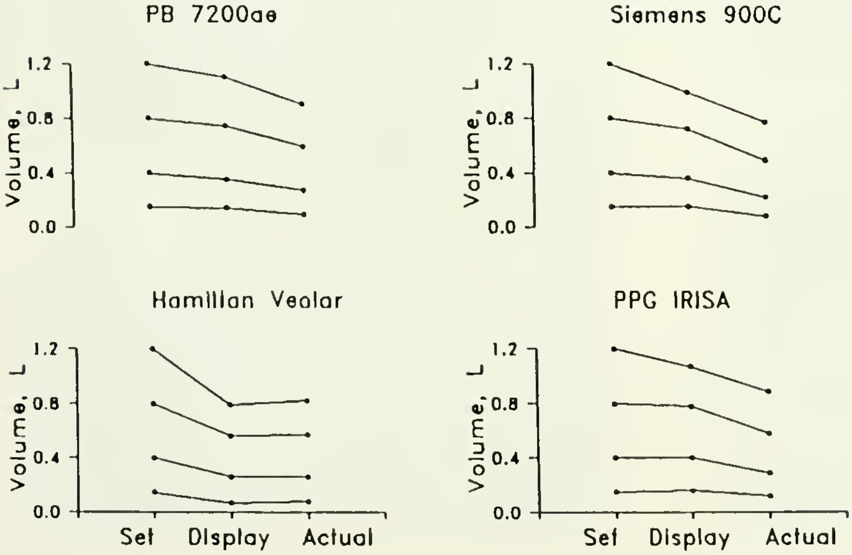

Fig. 11. Comparison of set volumes with the value displayed on a ventilator monitor and that actually measured with a pneumotachograph. Measurements were obtained with four different set volumes for four ventilators attached to a test lung with a compliance of 0.01 L/cm H_2O. The least discrepancy between displayed and actual volume was noted with the Hamilton Veolar ventilator, reflecting the use of a proximal site for measuring volume. (Based on data from Reference 61.)

imal site is also an option on the Hamilton Veolar and Amadeus ventilators); in the Siemens 900C ventilator, the sensor in the expiratory limb is responsible for the digital displays of P_{peak}, $P_{plateau}$, and $\overline{P}_{aw}$, while the sensor in the inspiratory limb is responsible for readings of PEEP, system pressure, and movement of the needle on the analog gauge (Bradford M Saunders, Siemens Life Support Systems, personal communication, 1992). A pressure sensor located on the inspiratory side of the circuit is affected by the resistance of the humidifier and circuit, resulting in an underestimation of peak inspiratory pressure and an overestimation of end-expiratory pressure. Although measuring pressure at the proximal airway is preferred on theoretical grounds, this location is associated with a number of limitations. Inaccurate measurements may result from exposure of the sensor to secretions and water condensation, or the tubing connecting the sensing port with the pressure transducer may have an unsatisfactory configuration, be excessive in length, or become kinked or disconnected. Consequently, many manufacturers prefer placing the sensor in the expiratory circuit to avoid these problems.

A distinction should be made between optimal location of sensors for capturing data to drive the ventilator machinery and the positioning preferred for monitoring purposes. For example, although the Hamilton ventilator measures flow at the proximal airway (which generates the digital display of tidal volume), this information is not employed in the internal operation of the machine; instead, the manufacturer prefers to rely on an internal, protected transducer to regulate the logic system of the machine (David Thompson, Hamilton Medical, personal communication, 1992).

Display of Monitored Data

The optimal manner of displaying monitored data in ventilator-supported patients deserves serious investigation. Older ventilators simply displayed data in terms of movement of a needle on a dial or a digital readout of calculated measurements. With the introduction of microprocessor-based ventilators, it has become possible to store large amounts of data for manipulation and display in various ways[62] (Table 4).

Table 4. Options for Displaying Monitored Data on Ventilators

- Movement of needle on a dial
- Digital readout
- Waveform over time
  - Raw signal
  - Filtered signal
  - Compressed over variable time periods
- Derived indices
- Trend plots
- Interpretation

It is very difficult to effectively assimilate long columns of numbers, and graphic representation is preferable. Although most data are reduced to calculated indices, some information may be better presented in waveform fashion. In this situation, employment of pattern recognition may yield a more rapid diagnosis than assessment of derived indices. Examples include the scallop seen in the pressure-time curve in a patient receiving an inadequate inspiratory flowrate (Fig. 1) or the curvilinear expiratory flow profile of the flow-volume curve in a patient with COPD (Fig. 6).

Clinical decisions are commonly based on trends rather than on isolated values of an index. Many questions related to trends are unanswered. How much data should be included in producing a trend? Should the data be smoothed, and if so, how much? How great a change in a trend is required before it can be considered significant? In attempting to answer the last question, the trend could be plotted on a background display of the normal range, or the microprocessor could calculate the 95% confidence limits for the data over a preceding time period and plot the incoming data on this background display. Finally, the voluminous amounts of data generated by sensors in a mechanical ventilator may produce information overload.[63] Again, the microprocessor could assist by interpreting the various indices and rendering a diagnostic opinion.

In Conclusion

Accurate measurements of pressure, flow, and volume are a fundamental requirement for the effective operation of mechanical ventilators and the minimization of iatrogenic problems. The accuracy of the pressure and flow sensors in various ventilators is largely unknown because few if any investigations of their accuracy have been published in peer-reviewed journals. Likewise, peer-reviewed studies are needed to determine the optimal location of each type of transducer for patient monitoring and to determine the preferred location of sensors used in the regulation of the mechanical components of the ventilator. In addition to the development of methods of recording accurate physiologic data, more research is required to determine the relative value of different derived indices in clinical decision making and the optimal format for displaying data. Considering the widespread use of mechanical ventilation, initiation of research to answer these questions is a matter of some urgency.

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What Derived Variables Should Be Monitored during Mechanical Ventilation?

John J Marini MD

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- **II. Mechanics**
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Introduction

Many variables are undeniably important for monitoring the patient receiving mechanical ventilation. In difficult cases, data relevant to gas exchange, hemodynamics, machine function, and lung and chest-wall properties are all helpful to consider. Information related to gas exchange and hemodynamics, although vital to integrate with other data, is seldom determined directly by the mechanical ventilator itself and will not be considered further in this review. Currently, the primary measurements made by the ventilator are airway pressure, flow, and ventilatory frequency. Certain spirometric variables computed from these direct measurements (such as tidal volume and minute ventilation) can be considered primary, as well. Esophageal pressure, although directly measured and essential to several calculations, is not routinely recorded or interfaced to the ventilator. From the rather confined set of primary measurements just listed, a number of variables can be derived that are

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Dr Marini is Professor of Medicine, University of Minnesota, and Director of Pulmonary Medicine and Critical Care, St Paul-Ramsey Medical Center, St Paul, Minnesota.

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Reprints: John J Marini MD, St Paul-Ramsey Med Ctr, 640 Jackson St, St Paul MN 55101-2595.

of value in specific clinical settings. Generally speaking, such information relates to the assessment of mechanics, patient effort and ventilatory reserve, muscle strength, synchrony of the cycling rhythms of the patient and machine, and the mechanical function of the ventilator itself. Each grouping of these derived variables will be considered separately.

Mechanics

The mechanics of the respiratory system are those properties that influence the pressure requirement resulting from the selection of tidal volume, frequency, and flow pattern. In my view, four such measurements can be considered essential in the evaluation of the ventilated patient with critical illness: resistance of the patient and the external circuit; compliance of the lung and chest wall; end-expiratory lung pressure (the sum of PEEP and auto-PEEP); and mean airway pressure, a measurement of greatest value during passive inflation. Three other measures of mechanics are desirable to monitor in selected circumstances but cannot be considered *essential* to the care of most patients. These are the static pressure-volume loop, the amount of gas that is spirometrically measurable during prolonged exhalation, and the absolute lung volume.

Resistance and Compliance

Resistance and compliance are the derived variables most commonly recorded by the practitioner. These calculations describe the impedance encountered by an applied pressure in driving gas to the alveolar level and in stretching the alveolus against the combined recoil of the lung and the chest wall. Accordingly, the relevant pressure differences across the airway and across the alveolus must be known. When only tracings of airway pressure and flow are available, resistance and compliance must be assessed under passive conditions. If the patient makes efforts during the attempted measurement, the total pressure applied across the entire respiratory system cannot be determined directly. When esophageal pressure (P_{es}) is available, the resistance (R_{aw}) and compliance (C_L) of the lung (inherently a passive structure) can be assessed, but the resistance and compliance of the chest wall remain undeterminable. P_{es} recorded during passive inflation allows the clinician to partition the pressure applied at the airway opening across the lung and chest wall and thereby to determine the individual contributions of those structures to total elastic recoil. During active inspiration, P_{es} also permits the computation of R_{aw} and C_L.

In the usual clinical setting, only an airway pressure tracing and a flow tracing are available. When the rate of airflow at end-expiration is known, as under constant-flow conditions, R_{aw} and C_L can be computed conveniently by imposing a pause lasting 0.3-2.5 seconds at end-inspiration. The appropriate pause length varies with the nature and severity of disease and the heterogeneity of ventilatory time constants among different lung regions.[1] The difference between the end-inspiratory dynamic and static pressures represents the pressure driving end-inspiratory flow between the airway opening and the alveolus. The difference between the *static* end-inspiratory and *static* end-expiratory airway pressures yields an estimate of the pressure change required to distend the alveolus by the tidal volume (V_T). This information can be used to estimate effective compliance (C_{eff}), as discussed later.

Effects of Lung Volume on Resistance and Compliance

Although this technique for partitioning the total applied airway pressure into its flow-resistive and elastic components is time-honored and simply implemented, it has recently been re-examined. At the bedside, it is seldom taken into account that the values of resistance and compliance computed in this way are influenced by the overall volume of the aerated lung. As an example, a patient who has undergone pneumonectomy will have a lower *absolute* compliance and higher *absolute* resistance than the same patient did before the lung was resected, even though the properties of the residual airway and the remaining lung tissue might be essentially unaffected by the procedure. If we are interested in resistance and compliance as indicators of the structural properties of the underlying tissue, it would be desirable to relate these numbers to the 'aeratable' lung volume (ALV). Unfortunately, it is

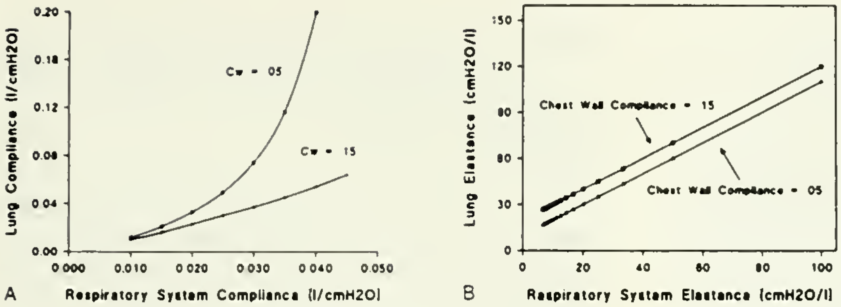

Fig. 1. Relationship between compliances and elastances of the respiratory system and lung. Note curvilinear relationship of measured respiratory system compliance to the lung compliance value of interest, especially when chest-wall compliance (C_W) is reduced. Conversely, for each C_W value a perfectly linear relationship of respiratory system elastance (1/C) to lung elastance exists.

difficult to measure *absolute* gas volumes at the bedside, and therefore such computations are seldom performed. Moreover, the interpretation of specific resistance is influenced by the homogeneity or heterogeneity of the parenchymal disorder. Diffuse pathology of lung tissue with a preserved number of patent conducting airways that link with a reduced number of aerated functional alveoli may result in less disparity between *absolute* and *specific* resistance calculations than highly regionalized atelectasis or air-space disease. (The latter tends to cause functional—if not anatomic—closure of airways and lung units.) Nonetheless, the potential influence of reduced ALV should be taken into account when *interpreting* values for resistance and compliance. During ARDS, the ALV may be less than one third of normal, due to cellular infiltration and edema.[2] Consequently, the computed compliance and resistance values, although clearly abnormal in this disease, may not reflect the true properties of the underlying tissue.[3]

Elastance

The reciprocal values of resistance and compliance are conductance (1/R) and elastance (1/C). Although there seems to be little inherent value in expressing the kinetic impedance properties of the respiratory system in terms of conductance, a better argument can be made for using elastance rather than compliance in the clinical setting. Unless a P_{es} recording is available, only static airway pressure is known, and the clinician can compute only the compliance of the entire respiratory system. Even then, passive inflation conditions must be assured. Therefore, the mechanical properties of the lung must be inferred from those of the lung and chest wall together. The compliance of the respiratory system is influenced not only by C_L—the usual variable of interest—but also by the compliance of the chest wall. Whereas elastances of lung and chest wall add in series to comprise the elastance of the respiratory system, the same does not hold true for compliance.[4] C_L bears a complex relationship to the compliance of the respiratory system, so that the alterations in chest-wall compliance so common in the critical care setting can seriously impact the relationship between the measurable respiratory system compliance and the lung compliance of actual interest (Fig. 1). Abnormal chest-wall compliance does not disturb the essentially linear relationship between *respiratory system elastance* and *lung elastance*, however; thus, such measurements more reliably track changes in the properties of the lung itself. Despite this rather compelling argument, compliance continues to be the index more commonly recorded.

Effects of Auto-PEEP and Gas Compression

Computations of C_L are strongly influenced by changes in auto-PEEP, a point first brought to clinical attention by Rossi and colleagues.[5] Thoracic compliance, normally considered to be the quotient of V_T and the difference between end-inspiratory static pressure and end-expiratory airway pressure, will be misleadingly low when auto-PEEP is unaccounted for. Although auto-PEEP's effect on compliance is well known, it is not commonly recognized that the magnitude of difference will increase as V_T declines (Fig. 2).

It is important to consider the compressible volume stored in the circuit and lung when C_L is com-

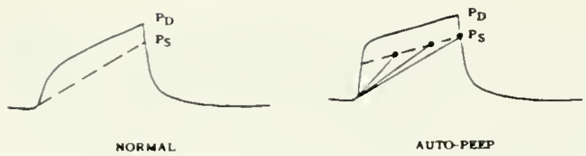

Fig. 2. Effect of auto-PEEP and tidal volume on measured elastance (and its inverse, compliance). During inflation with constant flow, the inspiratory airway pressure tracing obtained under passive conditions simulates a volume-pressure curve whose slope equals the true elastance of the respiratory system. If unaccounted for, auto-PEEP can radically increase the computed elastance and reduce compliance, especially at small tidal volumes. P_D and P_S are the peak dynamic and peak static values of tidal airway pressure, respectively.

puted from measurements of central airway pressure. The tidal volume effectively delivered to the lung must be corrected for gas compression as well as for any influence of auto-PEEP. Effective compliance can then be computed according to the formula

$$C_{eff} = \frac{V_T - (P_{stat} - PEEP)(compression\ factor)}{P_{stat} - P_{alv\ endex}}. \qquad [1]$$

where C_{eff} = effective compliance, V_T = tidal volume, P_{stat} = peak static (or "plateau") pressure, and $P_{alv\,endex}$ = total end-expiratory alveolar pressure (the sum of PEEP and auto-PEEP).

Static Pressure-Volume Curve

Although seldom computed or displayed, the static pressure-volume curve of the respiratory system can help determine both the heterogeneity of lung pathology and the optimal end-expiratory pressure needed to maximally recruit collapsed but uncompromised lung units. In patients with edematous lungs, the static inspiratory pressure-volume relationship is often biphasic, showing a distinct region of inflection (termed P_{flex}) that helps identify the point of near-maximum volume recruitment.[6] In most experimental and clinical studies, P_{flex} tends to occur in the pressure range of 7-12 cm H_2O and may well correspond to the "best PEEP" value determined by other methods.[6-8] Later in the course of ARDS, or when a sufficiently high PEEP has been applied, distinct P_{flex} points are less easily identified.

Dynamic and Static Evaluation of Respiratory System Mechanics

Static Measurements

The airway pressure tracing obtained under constant-flow conditions warrants close inspection, especially in the early inspiratory and end-inspiratory pause intervals. Traditionally, the pressure used to compute nonelastic impedance (resistance) during passive inflation with constant flow has been taken to be the difference between peak dynamic (P_D) and peak static (P_S, plateau) pressures. Yet, this difference comprises two subsegments (Fig. 3). When subtracted from P_D, the airway pressure corresponding to the point of inspiratory flow cessation (P_Z) may more accurately identify the pressure difference actually driving gas flow through the conducting airways. A resistance value computed with this pressure difference is sometimes called R_{min}. The drop in central airway pressure that occurs during the period of no *external* flow extending from P_Z to the "steady plateau or static pressure" value observed 1-2.5 seconds following airway occlusion (P_S) represents the pressure difference corresponding to internal gas redistribution between lung regions with different time constants (so-called pendelluft), as well as stress relaxation of the pulmonary and chest-wall tissues. P_Z is a value that should be carefully noted. Not only is P_Z important in computing R_{min}, but it is also a better reflection of the highest end-inspiratory alveolar pressure than P_S. In ARDS, P_Z and P_S generally

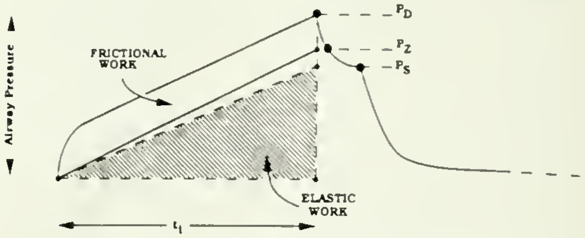

Fig. 3. Schematic diagram of the airway pressure tracing obtained during constant flow. As peak dynamic airway pressure (P_D) falls toward peak static pressure (P_S) during an end-inspiratory pause, the tracing passes through a point (P_Z) where gas stops flowing into the trachea. The P_D-P_Z difference, divided by the end-inspiratory flow, yields R_{min}. The P_D-P_S difference corresponds to R_{max} (see text).

differ by 10-20%—differences that might be meaningful in regard to the hazard of barotrauma.

The quotient of the total difference between P_D and P_S and end-inspiratory flow has been termed R_{max}.[9] It has been recently noted that R_{min} and R_{max} respond somewhat differently to changes in lung volume and inspiratory flowrate. As is pointed out by Bernasconi and colleagues,[10] patients with COPD and patients with ARDS, two groups believed to have nonhomogeneously distributed time constants, can have very significant differences for R_{min} and R_{max}, as well as for dynamic and static compliance.

Dynamic Measurements

The slope of the airway pressure curve during the second half of the constant-flow inflation cycle parallels the elastance of the respiratory system rather well. When this slope is extrapolated backward to the time of inflation onset, it intersects the pressure axis at a point that differs from the end-expiratory pressure baseline in a manner believed similar to the *end-inspiratory* pressure difference corresponding to the R_{max} calculation. Indeed, as was nicely shown by Gottfried, Rossi, and colleagues,[11] similar values for elastance and resistance of the respiratory system can be computed using either end-inspiratory "stop-flow" techniques or graphic methods during uninterrupted constant-flow inflation. This identity appears to hold, unless there is significant auto-PEEP or substantial volume recruitment during the early phase of tidal inflation. Finally, some estimate of R_{min} can also be achieved using constant-flow methodology by close analysis of the early inspiratory period.[3,12]

Expiratory Resistance

Expiratory resistance is another value commonly neglected in the patient receiving mechanical ventilation. Expiratory resistance almost invariably exceeds inspiratory resistance, even in normal subjects. With many ventilators in current use, expiratory valve resistance is often substantial, adding further to this phasic difference in resistance. Moreover, in certain patients (eg, those with COPD), dynamic airway compression markedly increases expiratory flow resistance. These considerations are important in the generation of auto-PEEP, particularly when minute ventilation requirements are high. Computation of expiratory valve resistance requires simultaneous recordings of the pressure at the airway opening and airflow. To compute total expiratory resistance, however, alveolar pressure (P_{alv}) itself must be estimated. P_{alv} can be measured indirectly from the pressure measured at the airway opening during occlusions performed one or more times during exhalation. The shape of the expiratory flow tracing may prove helpful in detecting the presence of dynamic airway compression when it demonstrates a biphasic contour or linear rather than exponential decay.[13] Another method for estimating P_{alv} during exhalation that does not involve interrupting airflow is to subtract the quotient of exhaled (spirometric) volume to compliance of the respiratory system from the P_S measured at the end of an end-inspiratory pause. This resultant pressure can then be used to estimate upstream alveolar pressure.

$$P_{alv} = P_S - \frac{V_{exh}}{C_{total}}. \qquad [2]$$

where P_{alv} = alveolar pressure, P_S = pressure at end-expiratory pause, V_{exh} = exhaled tidal volume, and C_{total} = total respiratory system compliance.

Resistance and Compliance during Spontaneous Breathing

During active breathing, the use of an esophageal balloon is invaluable for estimating the changes in intrapleural pressure associated with elastic and flow resistive impedance, as originally described by Rohrer[14] and Von Neergaard and Wirtz[15] more than a half century ago. Esophageal pressures corresponding to points of zero flow demarcating the phase of interest (inspiration or expiration) define the elastic pressure difference associated with the tidal volume change, thereby allowing computation of C_L (Fig. 4). The difference between the P_{es} tracing at the midpoints in the inspiratory and expiratory cycle and the line that joins end-inspiratory P_{es} and end-expiratory P_{es} defines the average gradient driving flow. During *passive* inflation, P_{es} can be used to partition total transrespiratory system pressure into its lung and chest-wall components. Under these conditions, the

P_{es} change represents the elastic pressure required to expand the chest wall by the tidal volume and therefore enables computation of its passive compliance.

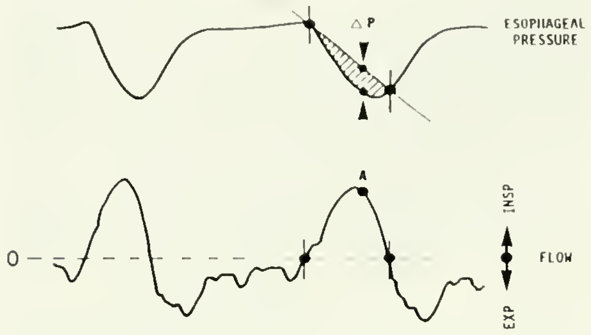

Fig. 4. Approximation of inspiratory resistance across the lung and attached apparatus during spontaneous breathing. The elastic recoil alveolar pressure can be approximated as esophageal pressure (P_{es}) at the points of zero flow that anchor the extremes of the tidal inflation cycle. The line drawn between them defines an elastic recoil line. The difference (ΔP) between P_{es} and that line approximates the component of P_{es} that drives the flow ($\dot{V}$) observed at Point A. The quotient $\Delta P/\dot{V}$ estimates airflow resistance.

Positioning of the esophageal balloon during active breathing is perhaps best undertaken by the method of Baydur and colleagues.[16] Here deflections of airway pressure (P_{aw}) and P_{es} are compared during gentle efforts against an occluded airway. The balloon can be considered to be positioned acceptably well when the P_{aw} and P_{es} deflections are similar. With the airway occluded, little *transpulmonary* pressure difference is developed at any point in the effort cycle, and, therefore, if P_{es} represents a true global average of intrapleural pressures, its deflections should not differ greatly from those recorded in the central airway. Balloon position should be re-evaluated after each major change in posture or position.

Mean Airway Pressure

During a passive breath, the mean airway pressure ($\bar{P}_{aw}$) recorded over the entire respiratory cycle provides valuable data often ignored in the clinical setting. Under these passive conditions, $\bar{P}_{aw}$ bears a close relationship to the average pressure within the alveolar compartment. Indeed, if flow-related pressure is dissipated in an identical fashion for both inspiration and expiration, $\bar{P}_{aw}$ will exactly equal $\bar{P}_{alv}$, as predicted by the following simplified formula:[17]

$$\bar{P}_{alv} = \bar{P}_{aw} + \frac{\dot{V}_E\,(R_E - R_I)}{60}, \qquad [3]$$

where $\dot{V}_E$ = minute ventilation, and R_I and R_E are measured values for the inspiratory and expiratory resistance of the segment that separates the point of P_{aw} measurement from the alveolus.

$\bar{P}_{aw}$ can be accurately estimated at any site in the external airway, provided that no significant asymmetrical resistance is interposed between the site of airway pressure measurement and the trachea (Fig. 5). Calculated values for R_I and R_E depend on the average flowrate pertaining to those phases of the ventilatory cycle. Because alterations of the inspiratory time fraction (t_i/t_{tot}) change the average flowrates of inspiration [$\dot{V}_E/(t_i/t_{tot})$] and expiration [$\dot{V}_E/(1 - t_i/t_{tot})$], any calculated difference between R_I and R_E (and therefore between $\bar{P}_{aw}$ and $\bar{P}_{alv}$) will be accentuated as t_i/t_{tot} deviates significantly from 0.5.

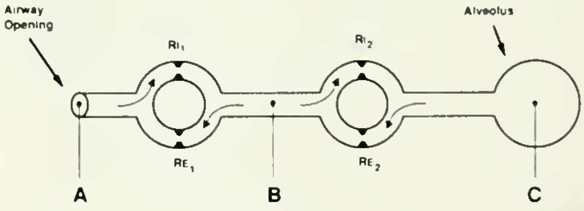

Fig. 5. Relationship of mean airway pressures at Points A and B to mean alveolar pressure (Point C). When inspiratory resistance (R_I) and expiratory resistance (R_E) values are equal, mean pressures anywhere along the conducting pathway are also equal. The mean pressure at B will exceed the mean pressure at A if $R_{E_1} > R_{I_1}$.

$\bar{P}_{aw}$ is determined by the need to overcome the flow-resistive and dynamic elastic components of impedance, as well as by pressures that are held continuously by any added PEEP or end-inspiratory pause. Under conditions of passive inflation, two desirable outcomes are linked to $\bar{P}_{aw}$: minute ventilation and arterial oxygenation. Unfortunately, increased $\bar{P}_{aw}$ may also result in impaired hemodynamics, fluid retention, or increased evidence of barotrauma.[17] The relationships of $\bar{P}_{aw}$ to $\dot{V}_E$ and arterial oxygenation help to explain why reducing $\dot{V}_E$ often impairs arterial oxygenation in the setting of acute respiratory failure, even when such an effect is not predicted by the alveolar gas equation. When $\dot{V}_E$ is reduced, arterial oxygenation may deteriorate, unless the corresponding reduction in $\bar{P}_{aw}$ is restored by an appropriate increase of t_i/t_{tot} or PEEP. Because of its ease of sampling and important physiologic implications, $\bar{P}_{aw}$ should be measured and displayed on all modern ventilators.

End-Expiratory Alveolar Pressure

End-expiratory alveolar pressure is the sum of the minimum pressure deliberately set at the airway opening (PEEP) and the pressure resulting from dynamic hyperinflation (auto-PEEP) (Fig. 6). The value of measuring end-expiratory alveolar pressure has been extensively documented in the recent literature.[5,18,19] Apart from its value in helping to explain changes in hemodynamics, alterations in arterial oxygenation, and artifacts in the monitoring of wedge pressure and compliance, recognition of auto-PEEP is essential in accounting for and reducing the work of breathing. A variety of techniques can be used to estimate auto-PEEP during passive inflation. These include end-expiratory port occlusion (either performed manually[18] or with the help of a device that temporarily diverts the inspiratory airflow from the mechanical ventilator—the Braschi valve[20,21]); the counterbalancing of auto-PEEP early in inspiration by applied airway pressure;[5] and the decline in end-inspiratory static pressure observed after frequency is transiently reduced to a low level. Another method for measuring auto-PEEP that does not involve interrupting the ventilatory cycle or pattern is to compute the difference between peak static pressure and the sum of PEEP and the quotient of tidal volume divided by compliance.

$$auto\text{-}PEEP = P_S - \left[PEEP + \frac{V_T}{C} \right]. \qquad [4]$$

This latter technique assumes that peak static pressure (P_S) is the sum of three components: PEEP, auto-PEEP, and the tidal elastic pressure (V_T/C). To compute compliance in this setting, it is best to use the end-inspiratory slope of the airway pressure ramp during constant flow, as previously discussed.[3]

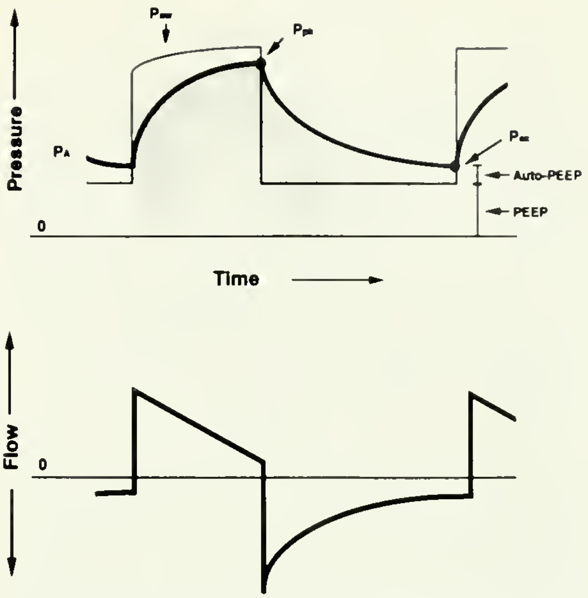

Fig. 6. Schematic pressure and flow tracings during volume-cycled, decelerating flow ventilation under passive conditions. On the top panel, alveolar pressure (P_A, thick line) rises to its peak (P_{pk}) and then decays exponentially to P_{ex}, the sum of PEEP and auto-PEEP.

Other methods for approximating auto-PEEP under passive conditions have also been described. One is to monitor total lung volume with impedance plethysmography, looking for the PEEP value that first causes an increase in end-expiratory volume.[22] A similar method is to observe the peak dynamic or static airway pressure as PEEP is added to the airway.[23] The highest value of added PEEP that fails to cause a proportional change in peak dynamic or static pressure has been suggested to be similar to the original level of auto-PEEP. Impedance plethysmography (or standard spirometry that compares tidal volume to total exhalable volume[24]) can also be used to detect the lung volume changes that occur as frequency is transiently slowed. During spontaneous efforts, auto-PEEP is perhaps best determined by the deflection of P_{es} required to stop end-expiratory flow.[25] In simultaneous comparisons of this value with that obtained by end-expiratory port occlusion, the esophageal counterbalancing technique tends to yield a lower value.

Effort and Reserve

Three sets of observations are essential in assessing effort and reserve: the minute ventilation ($\dot{V}_E$) requirement, the breathing pattern and the intensity of respiratory muscle activity, and the coordination among the muscles of the respiratory system. Because work output is a quadratic rather than a linear function of $\dot{V}_E$, $\dot{V}_E$ is an essential determinant of the effort expended, whatever the impedance to breathing or the patient's muscular reserves may be. Respiratory muscle activity and coordination give essential clues to the intensity of effort in relationship to capacity. For example, vigorous use of the sternocleidomastoid muscles or discoordination among the muscles of the rib cage and abdomen indicate an excessive stress.[26] Breathing frequency and V_T can be tracked continuously by the signal processing equipment of modern mechanical ventilators. In theory, a ventilator could combine such data into the frequency-to-V_T ratio proposed by Yang and Tobin to quantify rapid, shallow breathing.[27] This simple index recorded during a brief (1 min) trial of ventilator disconnection has been shown to have excellent predictive ability in judging the success or failure of a proposed weaning trial. To my knowledge, no ventilator currently available routinely presents this derived information.

Irregularity of the breathing rhythm, as indicated by wide fluctuations in frequency and/or V_T, also suggests serious overload of the respiratory muscles. Fortunately, for the purpose of monitoring, the changes just discussed in the pattern of respiratory muscle activation and in the breathing pattern tend to occur abruptly after stress is imposed or relieved, improving the clinical utility of such indicators.

A number of measures of effort and reserve that might be desirable and feasible to monitor using a mechanical ventilator are not currently considered essential to the scientific management of patients receiving ventilatory support. Among these are the inspiratory pressure-time product, the work of breathing, the "effort index," the power requirement, the maximum voluntary ventilation, the $P_{0.1}$ (occlusion pressure 100 ms into inspiration), and the mean inspiratory flowrate. During a period of passive inflation, the average inspiratory pressure recorded during constant inspiratory flow ($\bar{P}_I$) provides an index of the work per liter required from the ventilator for the specific tidal and inspiratory flowrate employed[28] (Fig. 7). The product of $\bar{P}_I$ and V_T yields the inspiratory work of breathing per tidal breath, and the product of $\bar{P}_I$ and $\dot{V}_E$ yields an estimate of the machine's ongoing power expenditure (work output per minute). Under unchanging passive conditions, $\bar{P}_I$ provides a convenient indicator of the impedance to breathing presented by the endotracheal tube and respiratory system. If the ventilator's tidal volume and flow settings can be matched to those of a spontaneously breathing subject, $\bar{P}_I$ can also estimate the patient's own pressure requirement with reasonable accuracy.[28] Such matching is difficult, however. Without an esophageal balloon in place, work requirement cannot be quantified; estimates rely on measurements of resistance, compliance, tidal volume, cycle timing, and flow. Of more value would be some measure of the patient's inspiratory pressure-time product, work of breathing, or power output during unassisted breathing.

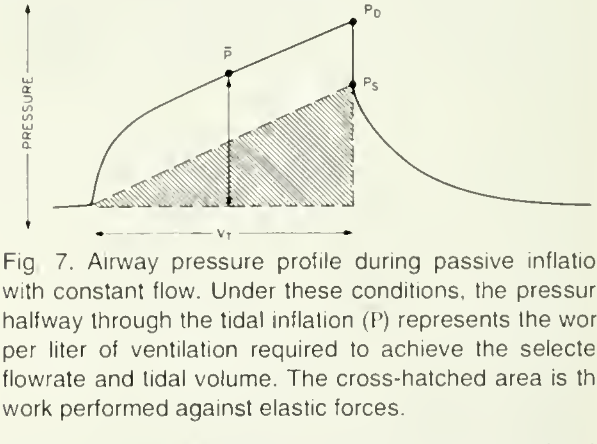

Fig 7. Airway pressure profile during passive inflation with constant flow. Under these conditions, the pressure halfway through the tidal inflation (P) represents the work per liter of ventilation required to achieve the selected flowrate and tidal volume. The cross-hatched area is the work performed against elastic forces.

During assisted machine cycles, the patient's work component can be gauged by comparing the machine's work output during controlled and assisted cycles of identical flow and V_T characteristics. Alternatively, patient effort can be assessed more directly by quantifying the inspiratory pressure-time product with an esophageal balloon. It is conceivable that a mechanical ventilator primed with data regarding R_I and C could keep track of the patient's inspiratory work of breathing by comparing the calculated value for total work accomplished to the ventilator's own contribution during triggered cycles. To my knowledge, such sophisticated estimates have not been incorporated or designed.

The occlusion pressure recorded 100 milliseconds after the initiation of a tidal effort ($P_{0.1}$) has been suggested as a useful indicator of patient effort and appears to correlate well with the ability of compromised patients to wean from mechanical ventilation.[29-31] The $P_{0.1}$ can be estimated rather closely by recording the pressure in the central airway during a brief, surreptitious occlusion of the inspiratory circuit. Indeed, the ventilator's own demand valve provides a delay that serves as a very brief occlusion during each breath, especially at low sensitivity settings.[32] Theoretically, the $P_{0.1}$ is a datapoint that could be gathered and displayed on command by the ventilator.

Comparison with the recorded $P_{0.1}$ before and after CO_2 stimulation may be the most effective method yet reported to predict weaning success or failure; the inability to increase $P_{0.1}$ after a CO_2 challenge indicates a patient at the limits of his/her reserve.[31]

Strength

The two measures of strength of most value during mechanical ventilation are the maximum inspiratory pressure (MIP) and the vital capacity (VC). In current practice, both MIP and VC are usually measured by encouraging the patient to make maximal efforts after disconnecting the mechanical ventilator. Several important features of such measurements should be taken into consideration, however. First, although the MIP measurement is usually made against an occluded airway, such values may be significantly higher than the maximum pressure that is possible to generate during a dynamic effort (Fig. 8). Second, inspirations against an occlusion should begin from a low lung volume and persist long enough to elicit maximal effort. Previous studies have demonstrated that 8-10 breaths or 20-25 seconds may be needed before MIPs are recorded and that the use of a valve that allows exhalation but prevents inhalation may maximize the recorded pressure values.[33]

Even when the patient is unconscious or poorly cooperative, the VC can be recorded by using a one-way valve to stack serial inspiratory efforts or to expel air sequentially from the expiratory reserve volume. Comparison of standard VC maneuvers with stacked VC maneuvers have shown remarkably good correlation, even in otherwise naive subjects.[34] Mechanical ventilators could certainly be adapted to facilitate MIP and VC measurements of this type.

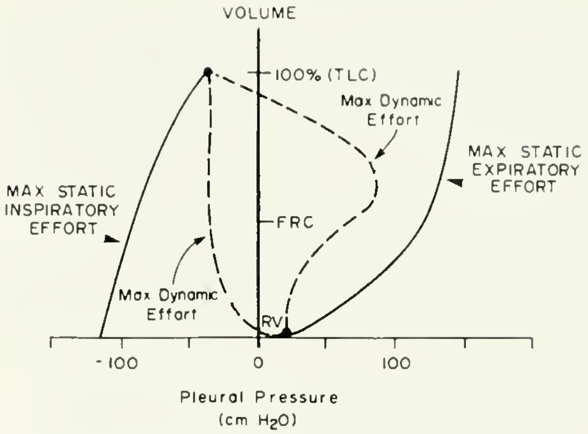

Fig. 8. Maximal inspiratory and expiratory pleural pressures in relation to lung volume. Note that the maximum pressures that can be generated during airflow (Max dynamic effort) are considerably lower than those achieved under static (occluded) conditions.

Ventilator and Circuit

The primary objectives of assessing the ventilator and circuit are to assure appropriate flow delivery in terms of magnitude and pattern and to assure that spontaneous breaths occur unimpeded by excessive circuit resistance. The primary means of assessing the performance of the ventilator and circuit with respect to these objectives are the indices provided by the airway pressure tracing and the flow delivery pattern.

The airway pressure tracing provides crucial data for assessing how well the ventilator is providing the desired flow pattern and how well the external circuitry succeeds in minimizing added impedance through the external circuit. It should be borne in mind that the inspiratory effort of a patient breathing spontaneously may be considerably greater than that suspected from examination of the central airway pressure tracing. Inspiratory effort through an endotracheal tube may be hidden from view unless pressure is recorded at the carinal end of the endotracheal tube. In the future, attempts may well be made by mechanical ventilators to sense pressure at the tracheal level or to compensate for endotracheal-tube resistance in other ways. At the present time, the level of pressure support needed to overcome endotracheal-tube resistance is a crude estimate, at best. Many tubes offer more resistance in practice than a clean endotracheal tube examined in vitro.[35] Moreover, at high frequency both the effectiveness of pressure support in achieving a given tidal volume and the contour of the applied pressure waveform itself deteriorate.

Patient-Ventilator Synchrony

Variations in peak airway pressures recorded during assist/control give a good clue as to the synchrony of the patient's inspiratory efforts with the duration of machine activation. Marked fluctuations in the observed peak airway pressure suggest that the patient's inspiratory efforts are asynchronous with the ventilator's flow delivery. Another key observation is a comparison of the patient's effort frequency to the triggered rate of the ventilator. Apart from extreme weakness or suppression of ventilatory drive, deflections of airway pressure that do not result in machine cycles strongly suggest that the triggering sensitivity of the ventilator is set inappropriately or that auto-PEEP is present. Close observation of the airway pressure and flow tracings can often reveal such asynchrony but, as in many other clinical situations, the best information is usually obtained by simultaneous observation of the patient's breathing pattern and the information recorded from the machine.

Summary

Without a careful definition, it is very difficult to propose a list of essential derived variables that should be monitored during mechanical ventilation. The list of essentials will vary not only with disease type and severity but also with the expertise of the operator in interpreting the data, and willingness to incorporate it into his/her surveillance and treatment plan. It can be cogently argued that the only variables of crucial significance to the vast majority of patients are the primary ones—airway pressure, flow, tidal volume, and minute ventilation. My own view is that end-inspiratory (P_D, P_S, and P_Z), end-expiratory (total PEEP), and mean airway pressures must be checked at frequent intervals, especially in ARDS. Partitioning of the total pressure into its flow-driving and elastance-counterbalancing components is always wise, whether or not resistance and compliance or elastance are formally calculated. Incremental changes in the pressure-volume relationship should be monitored whenever adjustments in PEEP or V_T are made. Ventilatory demand, strength, and power-reserve assessment are often instrumental in the care of the ventilator-dependent patient who presents as a weaning problem. The most valuable indicators of these include the $\dot{V}_E$, the maximum voluntary inspiratory pressure, and the frequency-to-tidal-volume ratio. Measurements of the work of breathing, $P_{0.1}$, and P_{es} should be reserved for unusually difficult clinical questions. Finally, the variability of the P_{aw} tracing yields valuable data regarding the synchrony of patient-ventilator interactions.

ACKNOWLEDGMENTS

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Essentials for Ventilator-Alarm Systems

Neil R MacIntyre MD and Sharon Day RRT

- **I. Background**
- **II. Alarm Sites**
  - **A. Site 1: Ventilator**
  - **B. Site 2: Patient-Ventilator**
  - **C. Site 3: Patient**
- **III. Alarm Events**
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Background

An alarm can be defined as "a signal that warns or alerts" or "a device that signals." One clinical definition of an alarm is "a mechanism that warns of potentially dangerous events."[1] The event may be a failure of the technology used to support a patient, or it may be a patient-generated phenomenon.

Modern mechanical-ventilation alarms derive from several decades of technical advances in mechanical ventilators. The earliest ventilators, both positive-pressure and negative-pressure models, lacked any alarms. The user had to rely on patient assessment and visual and audible signs of proper function (ie, chest movement and the sound of gas flow). As machine complexity increased, alarm capabilities followed to ensure that safe mechanical ventilatory support was indeed taking place. Later developments in alarm systems resulted in the capability to warn of important changes in patient physiology.

Alarm Sites

Current-generation (1992) mechanical ventilators have the capability to monitor events and actuate alarms in three conceptually different sites: (1) the ventilator itself, (2) the patient-ventilator interface, and (3) the patient.[1]

Dr MacIntyre is Medical Director, and Ms Day is Assistant Director, Respiratory Care, Duke University Medical Center, Durham, North Carolina.

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Reprints: Neil R MacIntyre MD, Duke University Medical Center, Respiratory Care Services, Box 3911, Durham NC 27710.

Site 1: Ventilator

Site 1 is in the ventilator and involves monitoring the machine's electrical and mechanical functions. Site-1 alarms are designed to alert the clinician to machine malfunction or failure. Potentially, from this site there can be monitoring of internal pressures, flows, timers, gas supply, and electronic function. Usually, however, only alarms for power loss, gas-supply loss, and machine malfunction are present.

Site 2: Patient-Ventilator Interface

Site 2 involves the interactions of the patient and the ventilator and uses monitors in the pneumatic ventilator circuit. Monitoring and alarm capabilities apply to circuit pressures (eg, peak, mean, plateau, and baseline 'airway' pressures), inhaled and exhaled volumes, delivered-gas flowrates, mechanical and spontaneous breaths, inspiratory and expiratory timing (I-E ratios), oxygen concentration, heat, and humidity.

Site-2 monitors and alarms provide the clinician with information about the patient as he or she interacts with the ventilator. Derived data and alarm activities may thus reflect either machine or patient changes or events. A low-exhaled-volume alarm, for example, may be generated by a circuit leak, or by a ventilator malfunction, or by a patient whose lung compliance is worsening or who is losing gas volume through a chest tube or around a poorly sealed endotracheal tube.

Site 3: Patient

Site 3 involves true patient monitors, such as devices to measure arterial oxygen saturation and end-tidal carbon dioxide concentrations. Although events detected by Site-3 alarms may possibly be related to the ventilator, these monitors are usually designed to detect patient events that are often independent of the mechanical ventilation systems. Site-3 monitors and alarms usually exist as standalone units, but they also may be part of a ventilator's monitoring package.

Alarm Events

The goal of mechanical-ventilator alarms is to warn of events.[1-4] An event in this context is any condition or occurrence that requires clinician awareness or action. Events related to mechanical ventilation can be conceptually divided into three levels or priorities.[1-8]

Level 1: Immediately Life-Threatening Events

Mechanical ventilators are life-support systems. Patients requiring mechanical ventilation may have little or no ability to breathe if the ventilator stops functioning. We should expect a Level-1 alarm to warn of every event in time for prompt correction to be made (Table 1). Because of the essential nature of this type of warning, alarms with redundant or overlapping function should be present. For instance, a failure to deliver gas should actuate a low-volume alarm, and, if practical, even a gas-exchange alarm (eg, capnography-triggered). This type of event should never be considered to have a 'mild' form: A serious failure exists every time it occurs. The alarms for Level-1 events are thus mandatory, redundant, and noncancelling. The goal is to provide virtually 100% reliability of the ventilator's life-support functions. Some machine failure is unavoidable,[3-5,9] but appropriate alarms can and must alert clinicians to technical breakdowns.

Table 1. Immediately Life-Threatening Ventilator Events (Level-1) and Related Monitors and Alarms

Event	Potential Monitors/Alarms
No gas delivery to patient	Pressure transducer, flow transducer, timing device, Site-1* monitors
Excessive gas delivery to patient	Pressure transducer, flow transducer, timing device, Site-1 monitors
Exhalation-valve failure	Pressure transducer, flow transducer, Site-1 monitors
Timing failure	Pressure transducer, flow transducer, timing monitor, Site-1 monitors
Electrical-power failure	Battery-powered alarm, Site-1 monitors

*Monitoring site within the ventilator.

When a Level-1 event occurs, the ventilator should not simply sound an alarm and shut down; rather, it should default to a condition in which the patient is, at the very least, not 'locked out' and is offered a source of fresh gas to breathe. The default-condition response to serious mechanical failure varies by ventilator manufacturer. It can range from an alarm message of "ventilator inoperative" and an opening of the patient circuit to atmosphere, to an alarm message and a backup minimum-ventilation mode.[9,10] Both responses are designed to alert clinicians to immediately replace the ventilator and to do the patient as little harm as possible during mechanical failure. A ventilator should also have several battery-backed alarms for Level-1 events in case of power-source failure.

Level 2: Ventilator Events Possibly Life-Threatening If Uncorrected

The events in this category can range from mild irregularities in the mechanical function of the ventilator to dangerous situations that, under certain circumstances, could threaten the patient's safety or life if left uncorrected for a prolonged period (Table 2). An example of a Level-2 event would be a circuit leak. A small leak (< 10 mL/breath) would not harm most adult patients, whereas a large leak (100 mL or more) might cause serious hypoventilation.

Alarms for these events are important and are present on nearly all modern ventilators. In fact, monitors and alarms for Level-1 events will often function for Level-2 events as well. However, redundancy for Level-2 events is usually not necessary.

Level 3: Patient Events

This type of event can have an important impact on the level of support given and on the pressure and volume consequences of that support. Level-3-alarm sites are usually in the ventilator-patient interface or, sometimes, on the patient (Table 3). Alarms for Level-1 and Level-2 events often function as alarms for Level-3 events as well. Redundancy, or separate alarms, however, should not be considered mandatory for Level-3 events.

Because patient status can change either abruptly or insidiously, the alarms for Level-3 events must be adjusted carefully for each patient, and they may need to be readjusted often to correlate with patient changes in order to remain accurate enough to warn of important problems.

Table 2. Ventilator Events Potentially Life-Threatening If Left Uncorrected (Level 2) and Related Monitors

Event	Potential Monitors for Alarms
Gas-blender failure	F_{IO_2} sensor
Loss of PEEP, or excessive PEEP	Pressure transducer
Autocycling	Timing monitor, flow transducer, CO_2 sensor
Circuit leak	Pressure transducer, flow transducer
Circuit partially occluded	Pressure transducer, flow transducer
Inappropriate I-E ratio	Timing monitor, flow transducer
Inappropriate heater/humidifier function	Temperature probe

Clinician-Alarm Interactions

Alarm Settings

Alarm setup involves selecting alarm settings that maximize sensitivity (ie, the percentage of true events that are detected) and specificity (ie, the per-

Table 3. Patient Events (Level 3) and Potential Monitors

Event	Potential Monitors for Alarms
Change in ventilatory drive (CNS, peripheral nerves, or muscle function)	Timing monitor, flow transducer, pressure transducer (in conjunction with airway occlusion), CO_2 analyzer
Change in compliance/resistance (air trapping, barotrauma)	Pressure transducer (in conjunction with inspiratory pauses), flow transducer
Auto-PEEP	Pressure transducer (in conjunction with airway occlusion), flow transducer

centage of false events that are ignored). However, a high sensitivity often leads to a loss of specificity and a high rate of false-positive alarms. A false-positive rate of 5%, for example, may be quite acceptable for a single alarm, but if a number of alarms with similar specificity are used together, the rate of false-positive alarms can rise dramatically.[11] This results in 'noise pollution' and a tendency for staff to ignore alarms. To minimize this, the clinician should limit the number of events that are covered by alarms and/or should individualize alarm ranges to maximize sensitivity for Level-1 events (at the cost of a loss of specificity)—and then strike a more even balance between sensitivity and specificity for Level-2 and Level-3 events.

Alarm Interpretation

Clinical skills are vital in determining the cause of an alarm. Several questions must be answered quickly: (1) Is the alarm reflecting a true event? (2) Is the event ventilator-related or patient-related, and how important is it (ie, a Level-1, -2, or -3 event)? (3) What response is necessary? Effectively answering these questions requires a high degree of clinical and technical expertise.

Alarm Packages

An alarm package for a mechanical ventilator can range from comprehensive to basic.

Comprehensive Alarm Packages

The addition of microprocessors to ventilators extends their alarm capabilities almost to infinity. The latest microprocessor ventilators provide a high degree of self-checking for both electromechanical and pneumatic systems. This can cause some arbitrary electronic tolerances to be so tight that they cause unnecessary alarms and service calls for conditions that do not cause any patient harm. This is a consequence of the facts that (1) there are no written standards for alarm-accuracy rates, and (2) mechanical failure is generally viewed to be the manufacturer and the user's liability and is not considered a reasonable risk for patients.

These comprehensive-approach systems can check electromechanical function, pneumatic function, and ventilator-patient interfaces every few milliseconds, and thus they create the potential for an enormously comprehensive alarm system. As noted above, however, this tremendous amount of sensitivity will usually result in a concomitant loss of specificity and consequent noise pollution. For this comprehensive approach to be clinically useful, therefore, microprocessors must be used to create 'smart' alarm packages that can be programmed to analyze information discriminately. Software programs can be designed to combine information, compare it to previous information, generate alarms, and even recommend corrective actions. Future software-design improvements may include variable alarm delays or multiple-breath-measurement averaging compared to an instant alarm or a breath-to-breath update on measurements. Microprocessors have the ability to link key settings with alarms and redundant alarms to prompt the user to make appropriate choices. An example of this would be a patient on pressure support, some of whose breaths have small tidal volumes. If the machine prompts the clinician to set appropriate minute-ventilation and respiratory-rate alarms, the tidal-volume alarm can safely be set lower, and this will eliminate an irritating, redundant alarm.

Basic Alarm Packages

At the other extreme is the minimal, or basic, approach, based on the concept that protecting the patient from ventilator malfunction is the only required goal. The alarm package is thus constructed to provide at least two alarms for Level-1 events and at least one alarm for Level-2 events. For example, such a package might include a gas-inlet alarm, a Site-1 malfunction alarm, a high-low pressure alarm, a high-low minute ventilation alarm, and a high-low temperature alarm (if a water humidifier is used).

The basic approach to the alarm package often is used on a ventilator without extensive microprocessor control. Costs are greatly reduced, and reliability may be increased secondary to the reduction in complexity. A reduction in user error may also accompany less complexity. While basic safety of the patient is provided, sophisticated automated responses usually are not possible with this approach. Extensive patient monitoring and alarm capabilities also do not usually exist with the basic approach.

In Conclusion

Manufacturers have accepted a charge to produce both reliable and redundant alarms to warn of almost any feasible ventilator failure. Alarms are mandatory only when applied to potentially life-threatening ventilator events. However, alarms are also important to warn of other ventilator or patient events that could affect clinical management.

Alarm settings require a balance of sensitivity and specificity. In the future, the simple concept that "if a variable can be measured, it should be monitored by an alarm" must be tempered by clinical judgment. When an alarm activates, the clinician must determine whether the problem is the ventilator or the patient (or both) and must be prepared to respond appropriately. Clinical skill and understanding of the alarm-system design are thus crucial.

Finally, while the basic approach to providing alarms for only Level-1 and Level-2 events with a simple system provides an appropriate safety standard, the capability to expand alarm systems to smart systems is a desirable goal for the future.

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Digital Electronic Communication between ICU Ventilators and Computers and Printers

Thomas D East PhD, W Hsueh-fen Young MS,
and Reed M Gardner PhD

I. Background
  A. Level A: Hardware for Digital Communication
  B. Level B: Handshaking between Devices
  C. Level C: Data Format
  D. Level D: Data Validation
  E. Level E: Representative Data
II. Current Status
III. Patient Outcome
IV. The Future
V. Summary and Recommendations

Introduction

When one ventures onto the exhibit-hall floor to 'kick the tires' of this year's latest and greatest ventilators, on almost all of them one sees communication ports designed to allow computers in the ventilator to 'talk' to external devices such as printers and other computers. If this feature is not included on a current model, then it usually is available as a low-cost option. The ventilator sales staff will eagerly point out this "valuable" feature and will be quick to imply that everyone needs this digital communication port in order to exist in the "modern computerized hospital environment." The clear impression given is that if you buy this elegant machine, all you need to do is make a simple connection between this port and any other computer, and all the necessary respiratory care data will be effectively and accurately transferred.

In reality, the current situation is more like buying a fancy new car, then finding that the radio receives only Japanese stations. If you are an electronics expert, you might be able to modify the radio to receive your local FM stations. It might be possible to buy a converter that fixes the problem; or you might accept the radio the way it is and learn Japanese. Most persons would just never use the radio, and that is exactly what happens with most of the digital communication ports on mechanical ventilators today. They sit unused.

The purpose of this paper is to provide a background on digital electronic communication and the problems encountered in interfacing a computer with a mechanical ventilator. The current state of the art and future directions are examined. Finally, three pivotal questions are addressed: (1) Is it essential to have a digital electronic communication

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Dr East is Associate Professor of Anesthesiology, Bioengineering, and Medical Informatics at the University of Utah, and Director of Informatics Research in the Pulmonary Division, LDS Hospital; Ms Young is a programmer with Intermountain Health Care (IHC); and Dr Gardner is Professor of Medical Informatics, University of Utah, and Director of Clinical Computing at LDS Hospital—Salt Lake City, Utah.

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Reprints: Thomas D East PhD, Pulmonary Division, LDS Hospital, 8th Ave and C St, Salt Lake City UT 84143.

port on a ventilator? (2) What impact do electronic data from a ventilator have on patient outcome? (3) If electronic communication is to be effective in the future, how should these interfaces be configured for mechanical ventilation?

Background

Digital communication of respiratory care information can be seen as having five hierarchical levels (A through E), as seen in Figure 1. The lowest level (A) is the basics of the hardware, the physical communication link. The intermediate levels are (B) handshaking between devices, (C) data format, and (D) validation of data. At the top level of this hierarchy is (E) the issue of whether data are representative. Effective communication on all these levels is essential if the system as a whole is to be beneficial to the clinician and to the patient.

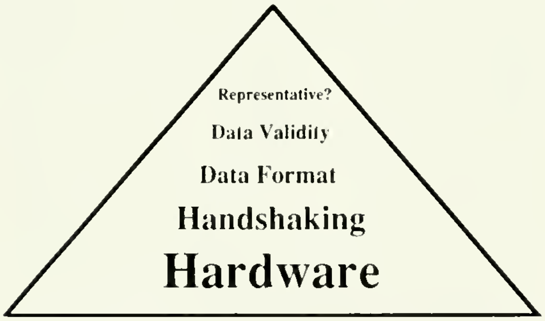

Fig. 1. The five levels of digital communication. Effective communication must exist at all five levels if electronic communication between the ventilator and the computer is to be helpful to the clinician.

Level A: Hardware for Digital Communication

The lowest level of our hierarchy is the hardware necessary to communicate digitally. Digital communication consists of representing numerals and letters by binary numbers. Each numeral and letter is assigned an ASCII (American Standard Code for Information Interchange) code number. These ASCII codes are represented by binary numbers that are made up of series of the numerals one (1) and zero (0). For example, the numeral 1 (actually considered a character for ASCII purposes) is assigned an ASCII value of 48, which when converted to base 2 or a binary number is 110000 ($110000 = 2^5 + 2^4$).

The most common digital communications on mechanical ventilators conform to a standard known as RS-232 (Fig. 2). The ASCII numbers are sent as a series of either seven or eight data bits (a 1 or a 0). Each bit is physically represented by a voltage (+3 to +25 V for a 1, and –3 to –25 V for a 0) that is present on the wires for a fixed time interval. The length of the fixed time interval for each bit is dependent on the baud rate (bits per second). Many different baud rates are used, from 100 to 19,200 bits per second. The RS-232 standard also defines 25 conductor cable wiring connections (Table 1) that can be used for RS-232 communication. A common mistake is to assume that if a device is claimed to have an RS-232 port, it will easily connect with any other RS-232 port on a computer or printer. The problem with the RS-232 standard is that it is very flexible—to the point of being close to being no standard at all. We like to refer to this phenomenon as the RS-232 myth.

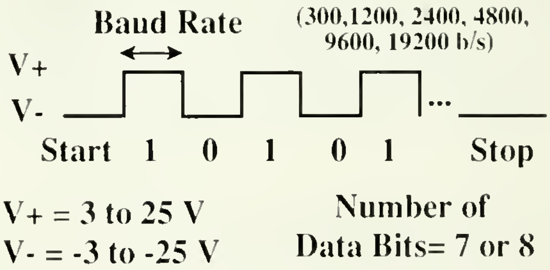
Baud Rate
(300,1200, 2400, 4800, 9600, 19200 b/s)
V+
V-
...
Start 1 0 1 0 1 Stop
V+ = 3 to 25 V
V- = -3 to -25 V
Number of Data Bits= 7 or 8

Fig. 2. RS-232 digital communication, the most common digital communication on mechanical ventilators. The ASCII numbers are sent as a series of either seven or eight data bits (a 1 or a 0). Each bit is physically represented by a voltage (+3 to +25 V for a 1, and –3 to –25 V for a 0) that is present on the wires for a fixed time interval. The length of the fixed time interval for each bit is dependent on the baud rate (bits per second). Many different baud rates are used, from 100 to 19,200 bits per second.

The following are common variables associated with the RS-232 standard: cable connector, connections (pin definitions), number of data bits, number of stop bits, baud rate, and parity. Parity is a bit used for error-checking. It must be defined whether

Table 1. RS-232 25-Pin D-Connector Pin Definitions

Pin No.	Symbol	Definition
1	FG	Frame Ground
2	TD	Transmitted Data*
3	RD	Received Data*
4	RTS	Request To Send
5	CTS	Clear To Send
6	DSR	Data Set Ready
7	SG	Signal Ground*
8	DCD	Data Carrier Detect
9		
10		
11	QM	Equalizer Mode
12	sDCD	Secondary DCD
13	sCTS	Secondary CTS
14	sTD	Secondary TD
15	TC	Transmitter Clock
16	sRD	Secondary RD
17	RD	Receiver Clock
18	DCR	Divided Clock Rcvr
19	sRTS	Secondary RTS
20	DTR	Data Terminal Ready
21	SQ	Signal Quality Detect
22	RI	Ring Indicator
23		Data Rate Selector
24	TC	Ext Trans Clock
25		Busy

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*Indicates essential connections.

Table 2. Digital Communications Port on Apple Macintosh Computer*

Pin	Definition
1	Handshake Out
2	Handshake In
3	Transmit Data –
4	Signal Ground
5	Receive Data –
6	Transmit Data +
7	General Purpose Input
8	Receive Data +

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*The connector is a DIN-8 microcircular connector.[1]

parity error-checking is used, and if it is used whether it is even or odd parity.

The electrical connector varies a great deal from device to device. The number of conductors in the cable can vary from 2 to 25. The types of connectors used include RJ-11 phone jack, DIN-8 round connector, 9-pin D-connector, 15-pin D-connector, and 25-pin D-connector. These connectors may be male or female. The definitions of the pins vary among manufacturers. For example, the connections for the digital communication port on the Apple Macintosh computer are shown in Table 2.[1] Compare Table 2 to Table 1, which is the RS-232 standard.

Even the definitions of "transmit" and "receive" are confusing. The manufacturer of a computer may define data going from his computer to the ventilator as "transmit" and data from the ventilator as "receive." The ventilator manufacturer, on the other hand, might define data going from the ventilator to the computer as "transmit." In such a situation, if the user connects "transmit" from both devices together, there is no communication because both devices are 'talking' and neither is 'listening.'

The number of data bits and stop bits, the baud rate, and parity information on a device can usually be set by the user, and one must match all these variables if communication is to occur. Some more sophisticated devices automatically adjust these variables to adapt to the device connected to them. Unfortunately, the truth is that even on the simplest level of digital communication, the hardware level, there is often a major communication gap.

Level B: Handshaking between Devices

The second level of digital communication is handshaking between devices. This can be viewed as the stoplight of electronic communication. The whole idea is to control traffic flow between the devices. If data are being sent to a device more rapidly than it can deal with them, the device needs a way to say "STOP! Wait until I am ready!" This is known as handshaking.

Two different general schemes exist: hardware handshaking and software handshaking. Hardware handshaking uses physical wires between the devices, such as data terminal ready (DTR), clear to send (CTS), and request to send (RTS) to control the flow of information. The disadvantage of hardware handshaking is that it requires larger connectors and more conductors in the cable. It can also be challenging to discover which connections are required for each device. The most popular technique is software handshaking, in which the only connections between the devices are transmit, receive, and signal ground. All the control of traffic is accomplished by use of special ASCII codes known as XON and XOFF, roughly equivalent to GO and STOP, respectively.

The handshaking must match on the two devices. If one device requires hardware handshaking and the other device supplies only software handshaking, there will be serious problems and unreliable communication. If hardware handshaking is used, it will be challenging to discover which connections are required for each device.

Level C: Data Format

The representation of the data is the third level of digital communication. The following is an example of the data stream from two different mechanical ventilators that are set up identically.

Ventilator 1: "Tidal volume = 350 mL"
Ventilator 2: "0.289"

The difference is that Ventilator 1 provides some verbal material to identify its value, and Ventilator 2 does not. In addition, Ventilator 2 uses liters rather than milliliters as the unit of measurement, and the tidal volume has been corrected for tubing compression-volume losses. Any computer interfaced with these two devices would have to deal with these supposedly identical data in two different manners. In order to communicate effectively, we must compare apples to apples and oranges to oranges. The sequence of variables and the format of the data stream must be carefully defined. All the units used should be the same, and any corrections should be consistent throughout. A frustrating fact has been that some medical device manufacturers have altered the structure of their stream of digital information from one version of their system software to another version. For example, a Puritan-Bennett (P-B) 7200 ventilator that has a pressure-control option installed will have a different data stream from a P-B 7200 that does not have that option. This third level of digital communication—data format—does not pose a tremendous problem if only one specific ventilator is to be interfaced with a computer. However, if more than one type of ventilator is used, accommodating all the different representations of data can be overwhelming.

Level D: Data Validation

The fourth level of digital communication is validation of the data. If the data sent from the ventilator are not valid, then it is impossible to transfer effective respiratory care information to the clinician. Validation involves checking all the data to make sure they are reasonable. For example, tidal volumes of 10 mL probably are not valid and should not be sent from the ventilator. This level of communication is essentially missing from all mechanical ventilators on the market as this paper is being written (early 1992).

A main reason that data are sent out by ventilators with no attempt being made to validate them is legal liability. Manufacturers are worried that if their devices make an interpretation of data, they can be potentially liable for missing data or bad data that in some way harm a patient. Although this is a very real concern, sending nonvalidated data may also be misleading and potentially cause harm. It seems that tradition has dictated that it is all right to display invalid information on meters and digital displays, and that therefore this is the safest legal path. We will never have successful electronic communication until we have solved this legal issue. There is no doubt that, in the long run, having high quality, validated data is in the patient's best interest.

Level E: Representative Data

The most important issue for medical decision making is whether the data are representative of the patient's state (Fig. 3). This is the highest level of digital communication. Only data that are truly representative should be sent from the ventilator. Figure 4 illustrates the problem. The raw data supplied from the ventilator at 10-second intervals have wide variability. There is also an immense quantity of information. For example, we obtained about 1,500,000 bytes (characters) of data from a P-B 7200 ventilator over a 24-hour period for one patient. This is equivalent to approximately one fourth the length of the Bible. This vast amount of data definitely produces information-overload for the clinician at the bedside. It certainly is not reasonable to just dump all these data on clinicians and expect them to use the information effectively.

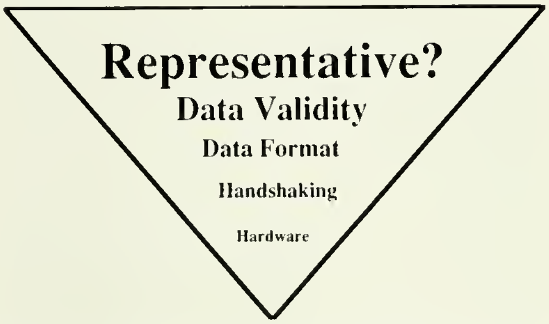

Fig. 3. The relative impact of the five levels of digital communication on medical decision making.

The data in Figure 4 were validated and represent actual patient events. Very few of these fluctuations are charted at present in the manually kept patient record. This raises questions: What is important to record? How often should data be recorded? What constitutes an artifact? How can you tell what is a significant event? We presented a collection of graphical raw data recordings to respiratory therapists, physicians, and nurses—and asked them to identify what they felt were important events that needed to be charted. Not surprisingly, the most common answer was to chart every 2 hours because this was the clinical practice. As we probed past this automatic response, we found that the answers varied widely. Nearly everyone agreed that all changes in ventilator settings should be charted. However, deciding which measured variables to record was more difficult. What were lacking were agreed-upon definitions of artifact and significant event.

In Figure 4, much of the data in the raw signal would be considered artifactual by most clinicians. But, again, how is artifact defined? One leading dictionary lists six different meanings, including these three: (1) "a handmade object, as a tool, or the remains of one, as a shard of pottery, characteristic of an earlier time or cultural stage, esp. such an object found at an archaeological excavation," (2) "a spurious observation or result arising from preparatory or investigative procedures," and (3) "any feature that is not naturally present but is a product of an extrinsic agent, method, or the like."[2] Whereas the first of these definitions is probably the most familiar to the layperson, the second and third definitions are nearer the sense of artifact as it is used in describing or discussing the phenomenon in digital communication. Still, differences exist in

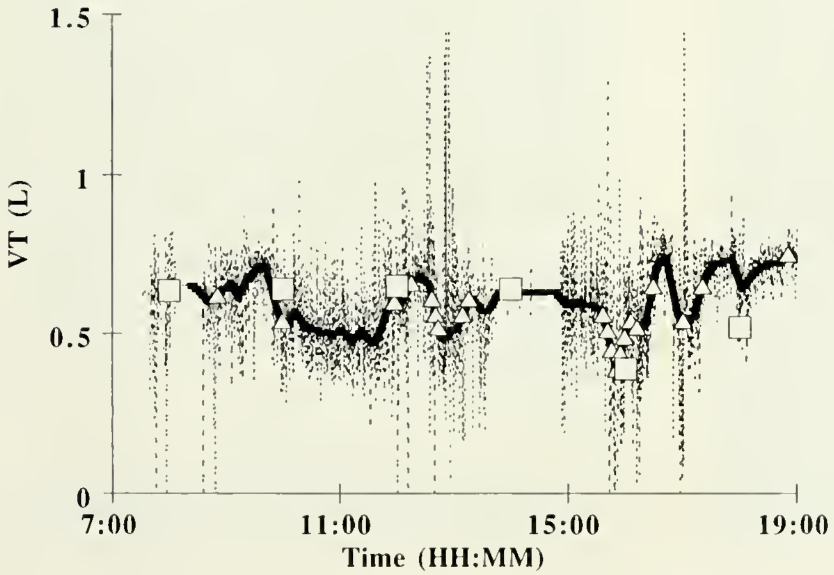

Fig. 4. An example of tidal-volume data collected from a ventilator at 10-second intervals. The effect of filtering with a LOESS filter is shown, and only the significant events are stored in the patient record. Raw data are indicated by verticle dashed lines, filtered data (LOESS filter) by heavy black line. Δ = significant events: ❑ = manually charted.

use of the term. One of the issues is the perspective of the observer. For example, an engineer designing a ventilator would consider an artifact to be anything that made the measured variable inaccurate, whereas a clinician would also include in his definition of artifact those values that were accurately measured but did not represent the true state of the patient.

The definition of what constitutes an artifact varies widely among clinicians. When asked for a specific definition, the clinician will typically ask about the specifics of the case and patient history. If this is any indication, then the definition of artifact must depend on the disease-patient complex and will vary not only among individual patients but also throughout the course of the disease. If this is true, then a clear definition of artifact may be very difficult to settle upon. Certainly, as mentioned above, a universally satisfactory and useful definition of artifact is lacking, and agreement may be as precious and difficult to find as artifacts from ancient Egyptian tombs.

Similar problems exist with definitions of a significant event. The problem that we encountered with defining a significant event was the end point chosen. If one chooses patient outcome as the end point, then only events that affect patient outcome are considered significant. Obviously, patient outcome is far too extreme an end point. There are no data in the literature to support a definition of what events have significant impact on patient outcome. What it boils down to is an educated guess as to what amount of change in a particular variable could potentially affect the patient significantly. An argument could be made for adjustable definitions of significance, depending on the patient's status. For example, the critically ill patient with the adult respiratory distress syndrome might be exquisitely sensitive to changes in mean airway pressure, whereas the average postsurgical patient does well no matter what the mean airway pressure is.

A different perspective on this issue is the legal one. We have sought our lawyers' opinions on how often we need to collect data to have a good legal record. The answer was circuitous, at best. They implied that we should collect data at the interval proven to be adequate. Inasmuch as an adequate interval has never been specified or proven, the next best thing would be to collect data at the same rate that everyone else does. The lawyers added the caveat that respiratory care practitioners should collect only data that they are prepared to act upon. Fetal monitoring is a good example of a situation in which it has been easy to collect large amounts of data, but if the obstetrician has not acted upon the data, he has been found to be liable. In some ways, from a legal perspective, if we are not prepared to act upon data more frequently than every 2 hours, it may be better to 'stick our heads in the sand' and pretend that nothing happens between those 2-hour ventilator checks.

One way to deal with definitions of artifact and significant event is to force the clinician at the bedside to make the decision. This is what is currently being done in many ICUs that have computer systems interfaced with ventilators. All data from the ventilators are collected at fixed intervals and displayed, either in graphs or in tabular form. The clinician is asked to retrospectively pick the valid, artifact-free, significant events for charting. We duplicated this procedure in our study by asking the clinicians to circle the points in the raw data that they felt should be charted. There was good agreement in the points chosen for ventilator settings; however, the measured datapoints chosen by each individual varied widely. It is understandable from a legal perspective that manufacturers do not want to be involved in making the decision as to what is artifact; however, it is unrealistic to expect a person at the bedside, who was not in the room when the data were generated, to retrospectively pick out the 'good' data from amongst all the noise. A slightly different version of this technique is to store automatically acquired data when someone in the room signals, by pressing a key, that the patient is in a representative state. This works well for the periods when someone is at the bedside; however, what should be done with all the data from times when no one is in the room? Should they just be ignored? Much of the research needed to answer these difficult questions remains to be done. No ventilators currently make any attempt to send only "representative" data.

Current Status

Digital communication in most modern ICU ventilators is provided at Level A (hardware) and

Level B (handshaking between devices). However, it is not often easy to accomplish even these levels. No standard exists for representation of data (Level C), and no ventilator vendors currently provide any support for data validation and checking representativeness, the two highest levels of digital communication.

Several systems are commercially available to interface with selected ventilators. Puritan-Bennett's Clinivision product interfaces directly with their 7200 ventilator. Various ICU computer systems have developed interfaces for the Puritan-Bennett 7200, the Siemens 900C and 990 Servo computer module, the Hamilton Amadeus and Veolar, the Bear 5, and other ventilators that provide digital communication ports. These are typically custom interfaces that are matched to specific ventilators. These custom interfaces can be expensive and difficult to maintain. This means that if you happen to have a computer system in your hospital and want to connect your ventilators to it, most likely you will have to spend a great deal of time and money to do so. With only about 20 new installations of ICU computer systems in 1991 in the more than 5,000 ICUs in the United States,[3] a lot of ICUs that use modern ventilators do not have computer systems. Many ventilators, therefore, have idle digital communication ports.

In some research systems, ventilators have been successfully interfaced with computers. Shabot et al at Cedars-Sinai in Los Angeles have interfaced their Hewlett-Packard ICU computer with the P-B 7200 ventilator.[4,5] In that system, data are sent from the ventilator only when the clinician at the bedside pushes a button or when a ventilator setting has been changed. Our group has set up research systems at LDS Hospital in Salt Lake City that interface computers with the Siemens 900C and 900i ventilators,[6-9] as well as with the Hamilton Amadeus and Puritan-Bennett 7200 ventilators.

To facilitate data acquisition from a wide variety of medical devices, a standardized medical information bus (MIB) has been proposed.[4] The MIB provides a local area network (LAN) around the patient that can be interfaced with all bedside devices and that allows data from each device to be stored in a central database in a standard format.[10-13] The MIB is being standardized by the Institute of Electrical and Electronic Engineers (IEEE, New York City NY) so that all hospitals and vendors can have a common data format and so that their computers can easily communicate with many bedside devices.[4] The MIB handles issues unique to medical data communications, such as automatic recognition of new devices placed at the bedside, automatic reconfiguration of the network, and association of a device with a particular patient's bedside.[4] Unfortunately, the currently proposed MIB standard does not include standards for digital communication at Levels D and E (artifact rejection and significant-event identification).[14,15] Comparing Figures 1 and 3, it is ironic that the largest amount of effort has been spent on standardizing digital communication at Levels A and B (hardware and handshaking), which are the least important to medical decision making.

A preliminary version of the MIB was installed at the 520-bed LDS Hospital in 1986 and was connected to the HELP system.[14-17] The HELP (Health Evaluation through Logical Processing) hospital information system,[18-22] which has been developed over a 30-year period, runs on a system of 12 computer fault-tolerant processors in tandem, using the Guardian Operating System. The system is fault-tolerant in that no one system problem is sufficient to halt system operation. This feature provides the system with excellent availability (it is up 99.75% of the time). Program files and patient data are stored on 14 disk drives. The 8 drives currently used for clinical purposes store 2.4 gigabytes of data, while the 6 drives used for research hold 8.8 gigabytes.

The clinical drives are mirrored (ie, two drives hold the same data), virtually eliminating the possibility of data loss by hardware failure. When accessing data from one of the mirrored drives, the system retrieves the data from the drive that has its 'read head' closest to the data, which minimizes data-retrieval time. Eighteen Charles River Data Systems (CRDS), UNIX-based minicomputers are interfaced with the HELP system. The CRDS machines serve as multiplexes and preprocessors for terminals on the nursing divisions, in Surgery, in the Pulmonary Division, and in the Medical Informatics Department. A total of 1,100 terminals and 200 laser printers are currently active throughout the hospital. About half of these are connected directly to the tandem computer; the other half are connected via CRDS machines. All beds are fully computerized and have terminals at each bedside as well as at the nursing station. A version of the MIB links many of the medical devices in the ICUs directly to the HELP system (Fig. 5).

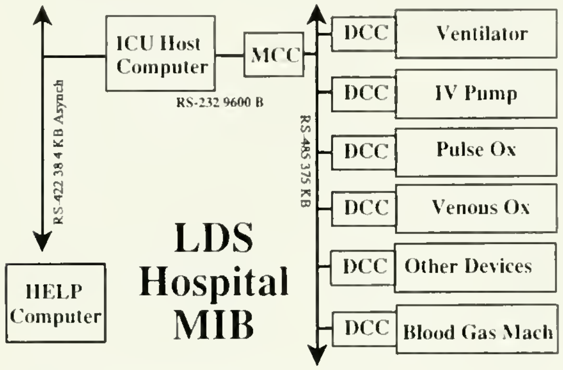

Fig. 5. The medical information bus (MIB) at LDS Hospital. A bedside network connecting many medical devices is in turn connected to a local network in the ICU and to the hospital information system. MCC = Master Communications Controller; DCC = Device Communications Controller.

An MIB interface for ventilators has been constructed at the LDS Hospital.[9] We recently completed studies investigating techniques for identifying artifacts and significant events, in which data were collected for 617 hours from 10 patients ventilated by Puritan-Bennett 7200 ventilators. Data from the ventilators were sampled at 10-second intervals and stored in a research database. This large database was then used to examine six different filters designed to eliminate artifact: (1) a moving-average filter, (2) a moving-median filter, (3 and 4) two different exponentially weighted moving-average filters, (5) a LOESS (a robust locally weighted regression technique) filter,[23] and (6) a moving-LOESS filter. Significant events were identified as values above a defined threshold (Table 3) for a critical period of time (T_{crit}). Two different T_{crit} times were used: 1 and 3 minutes. The output from each of these algorithms (a combination of a filter and a significant-event definition) was compared with the concurrent manually charted data in the HELP system.

Table 3. Definitions of Significant Events

MIB Variable	Thresholds
Mode setting	Every change lasting > 3 minutes
VR* setting	Every change lasting > 3 minutes
V_T setting	Every change lasting > 3 minutes
Peak inspiratory flow	Every change lasting > 3 minutes
F_{IO_2} setting	Every change lasting > 3 minutes
Trigger sensitivity setting	Every change lasting > 3 minutes
PEEP setting	Every change lasting > 3 minutes
Plateau setting	Every change lasting > 3 minutes
Pressure support setting	Every change lasting > 3 minutes
Flow-by setting	Every change lasting > 3 minutes
Flow-by sensitivity setting	Every change lasting > 3 minutes
P_{peak}	(Change > 10 cm H_2O) *and* (3 < P_{peak} < 120 cm H_2O) *and* 1 min after ventilator mode was changed
P_{aw}	(Change > 5 cm H_2O) *and* (3 < P_{aw} < 120 cm H_2O) *and* 1 min after ventilator mode was changed
$P_{plateau}$	(change > 5 cm H_2O) *and* (3 < $P_{plateau}$ < 120 cm H_2O) *and* 1 min after ventilator mode was changed
I:E	(% change > 25%) *and* 1 min after ventilator mode was changed
Spontaneous V_T	(% change > 10%) *and* (change > 100 mL) *and* (100 < spontaneous V_T < 2500 mL) *and* 1 min after ventilator mode was changed
Machine V_T	(% change > 10%) *and* (change > 50 mL) *and* (100 < machine V_T < 2500 mL) *and* 1 min after ventilator mode was changed
Spontaneous VR	(% change > 10%) *and* (change > 5 breaths/min) *and* (0.5 < spontaneous VR < 70 breaths/min) *and* 1 min after ventilator mode was changed
Machine VR	(% change > 10%) *and* (change > 2 breaths/min) *and* (0.5 < machine VR < 70 breaths/min) *and* 1 min after ventilator mode was changed

*VR = ventilatory rate.

Some differences existed between ventilator settings charted by respiratory care practitioners (RCPs) and those charted by the MIB. The error rate for manual charting of ventilator settings was 3%. By careful screening, we found that most of the difference came from (1) RCPs' back-charting with the wrong time-stamp, and (2) the time delay of the automated charting algorithms (1 minute for

T_{crit} = 1 min, and 3 minutes for T_{crit} = 3 min). RCPs tended to enter data and to stamp the time at which they *thought* the events occurred. Occasionally this time-stamp was in error. The error rate for manual charting of ventilator settings was reduced to 1% if all errors caused by back-charting were neglected.

Figure 4 shows an example of the tidal-volume data collected during this study. The raw data contained a lot of 'noise' and artifact. In general, all the filtering algorithms helped to reduce artifact, with the LOESS filter performing best, although it has the disadvantage that it requires much more computer time than a simple moving-median filter. The moving-median filter seemed to be the best choice because it did not follow transient events and was relatively simple to implement. There were large differences between numbers of events deemed "significant" by the algorithm and those charted manually. Two main differences were observed: (1) the RCPs did not chart what occurred when they were out of the room, and (2) when they did chart, they typically just 'took a snapshot' for a few seconds as they were working on the ventilator, which may not have been representative of the patient in the larger context.

Patient Outcome

There are few data on the impact of an automated respiratory care data-acquisition system on patient outcome. In our recent study (unpublished), we found that we could reduce ventilator-setting charting errors from about 3% to nearly zero. For measured variables, the automated charting found significant events that had previously been undetected. However, there are no data about what impact these results might have on patient care. Automation of other areas of the patient record has been shown to improve the quality of the data and to reduce the amount of time spent on charting;[24] Andrews et al reported an 18% increase in respiratory care department productivity with use of a computerized charting system.[25] However, Bradshaw et al showed that nurses' time in direct patient contact had decreased with use of computer-based data entry in the ICU.[26] Perhaps part of the problem is that computerized systems to chart respiratory care data are expensive, and one way to justify them is by a reduced requirement for employees. If these systems do save the clinician time but there now are fewer clinicians, then the net time spent with the patient may be the same or less. In general, it is assumed by most that higher quality, more timely charting of respiratory care data would improve the quality of care; however, this remains to be proven.

The Future

If digital electronic communication with mechanical ventilators is to become a routine part of clinical care, we must standardize all five levels of digital communication with these devices. A standard, such as the MIB, must be adopted to make it easy to physically connect the devices. In addition, we need more research into the elusive definitions of artifact and significant events. In the next 10 years, the respiratory care community must take an active part in this process of standardization. Without clinical input, the standardization process is doomed to failure from the beginning. Our vision is that one day, connecting your ventilator to your computer will be as simple as plugging in a telephone, and that the data will be valid and representative of the patient's true condition.

Summary and Recommendations

Although many modern ICU ventilators offer the option of electronic communication, most of these systems are not used because there is a huge communication gap between the ventilator and the computer it might be connected to. When such systems are now used, a large part of what is communicated is artifactual and misleading. We need to overcome both legal and knowledge barriers in the effort to provide seamless communication between ventilators and computers. With regard to the specific issues raised in this paper, here are our answers.

Issue #1: Is it essential to have a digital electronic communication port on an ICU ventilator?

Answer: No, it is not essential. The purpose of the mechanical ventilator is to support pulmonary ventilation by supplying gas and pressure. There is no vital role for digital communication in the gas-delivery function of the ventilator; however, in the future it will be essential to have effective electronic communication in order to guarantee accurate and timely charting.

Issue #2: What impact does electronic communication between a ventilator and a computer have on patient outcome?

Answer: Our preliminary data show that electronic communication can reduce the number of charting errors and can improve the timeliness of data entry. However, there is little evidence, other than anecdotal, that this has any impact on patient outcome. Automated charting has been shown to reduce the time spent on charting.[24] This time-savings could be used to increase time spent in direct patient care, but there is no conclusive evidence that this occurs. In fact, one report on computerized charting systems indicates that the result is less time spent in direct patient care.[26]

Issue #3: If electronic communication is to be effective in the future, how should these interfaces be configured for mechanical ventilation?

Answer: We recommend an optimal algorithm for automated respiratory care charting that has been suggested.[14]

- Sampling frequency: Sample data from the ventilator every 10 seconds.
- Ventilator-setting changes: Report every new setting if change lasts more than 3 minutes.
- Measured respiratory care data:
    - Filter raw MIB-collected data with a 3-minute moving-median filter.
    - Report one filtered value every hour for each variable.
    - In addition, use a threshold table (Table 3) to define significant events.
    - Report changes that remain above threshold more than 3 minutes.
    - Report all measured respiratory-care data 1 minute following any ventilator-mode changes.

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What Constitutes an Order for Mechanical Ventilation, and Who Should Give The Order?

David J Pierson MD

I. Introduction
II. Requirements for Successful Mechanical Ventilation
III. What Needs To Be Decided?
IV. Who Should Decide?
V. Ventilator Settings or Outcomes?
VI. Protocol-Driven Ventilator Management
VII. Conclusions and Recommendations

Introduction

The organizers of this conference included a discussion of ventilator orders, despite the fact that this subject does not at first seem necessary to a consideration of the essential features and descriptions of the machines themselves. Such a decision was wise because of the increasing complexity and enhanced mechanical capabilities of today's ventilators, and their potential to cause unnecessary patient discomfort, if not life-threatening harm. To my knowledge, however, there are no previous generally available publications on ventilator orders, in contrast to the extensive literature on current ventilator modes and their clinical application.[1-9]

Nonetheless, the questions posed in the title of this paper must be answered each time a patient is placed on a ventilator. Lacking readily available data or the advice of experts, I first review several factors that affect how one orders and carries out mechanical ventilation, discuss the implications of these factors for who should be giving such orders and carrying out such management, and then develop several recommendations based on the needs of effective, safe patient management in the context of today's high-capability ventilators.

Requirements for Successful Mechanical Ventilation

Successful application of ventilatory support is a complex, integrated process extending far beyond the selection of initial ventilator settings (Table 1). Management of a patient with severe chronic obstructive pulmonary disease (COPD) following a lobectomy for lung cancer is very different from the care of a previously healthy victim of multiple trauma who has developed multiple organ systems dysfunction and the adult respiratory distress syndrome (ARDS). Even in a given clinical setting, physicians usually have several quite different management approaches from which to choose—eg, whether to minimize lung water or maximize peripheral oxygen delivery in the second patient just mentioned. Thus, it is important that ventilator management begin with accurate patient assessment and a clear overall treatment plan, as shown in the table.

Dr Pierson is Medical Director, Respiratory Care Department, Harborview Medical Center, and Professor of Medicine, University of Washington—Seattle, Washington.

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Reprints: David J Pierson MD, Respiratory Care Department, Harborview Medical Center, ZA-62, 325 Ninth Ave, Seattle WA 98104.

Table 1. Requirements for Successful Ventilator Management

- Appropriate diagnosis and patient assessment
- Presence of a consistent, overall treatment plan
- The ventilator
    - Capabilities
        - Design features
        - Appropriateness for clinical setting
    - Condition
        - Setup and readiness for use
        - Maintenance
        - Repair
- Orders for ventilator management
    - Decision making: How ventilator is to be used
    - Communication: Instructions to users
- Ventilator operation
    - Implementation of orders by users
- Documentation (medical, financial, legal)
    - Intent: Orders for ventilator management
    - Actual use: Charting during ventilator operation
- Clinical assessment and monitoring
    - Physiologic effects of ventilator management
    - Patient outcomes

Currently available ventilators vary greatly in capability and suitability for different clinical situations. Use of an inexpensive home-care ventilator in the intensive care unit (ICU) might be appropriate for a patient with stable Guillain-Barré syndrome, but would be inappropriate in weaning a patient with COPD via synchronized intermittent mandatory ventilation (SIMV) after a prolonged bout of acute respiratory failure. Similarly, a top-of-the-line, $30,000 ICU ventilator would provide several advantages in managing a patient with severe ARDS, whose total respiratory system compliance was 15 mL/cm H_2O and minute ventilation 25 L/min, but might be hard to justify economically for routine short-term postoperative management of patients with normal lungs. Correct setup, maintenance, and repair of the ventilator are also of obvious importance whatever its clinical application.

Orders for mechanical ventilation reflect the clinician's decision-making process and designate how the ventilator is to be used. The decisions must also be communicated clearly and precisely via the ventilator order, both for those actually operating the ventilator and for others involved in the patient's care.

Documentation has become increasingly important in health care, and nowhere is this more evident than in the ICU. In addition to the traditional function of creating a medical record for current and future providers of the patient's care, the clinician's documentation now serves as the basis for reimbursement and other financial functions, and it is on the quality and quantity of documentation that the outcome of any legal action related to ventilator management will rest. Both intent (eg, the ventilator orders) and actual use (as reflected by charting during ventilator operation) are components of the documentation of ventilator care.

Finally, successful management depends on appropriate assessment and monitoring of the patient, not just the machinery. This includes both physiology, acute pathology, and more global outcome measures.

What Needs To Be Decided?

Ventilator orders require first that decisions be made about how the ventilator is to be used in patient management; these decisions must be communicated to others participating in the patient's care; and the whole process needs to be documented as described above. What decisions need to be included in the orders? Especially in critically ill patients with severe respiratory dysfunction, ventilator management is an extremely complex operation, made increasingly more so with each new advance in ventilator design and with the introduction of each new ventilator mode.

This complexity can be appreciated by comparing the ventilators clinicians use today with those employed for the same purposes a generation ago. In the early 1970s, volume-limited ventilation was still largely unavailable in many institutions, and most patients requiring ventilatory support were managed with pressure-limited machines such as the Bird Mark 14 and the Bennett PR-2.

Table 2 lists the different functions that could be included in ventilator orders using the PR-2. Today's ICU ventilators are vastly more complex, as illustrated in Table 3 for the Siemens Servo 900C. In contrast to the 5 settings and single alarm of the PR-2, the 900C has 18 controls and settings, plus 14 monitors and alarms that can be adjusted by the clinician. In addition, circuit compressible volume plus humidification and heating of the inspired gas mixture have to be accounted for and adjusted. Sel-

Table 2. Ventilator Orders 1972: Components in the Bennett PR-2

- Controls and Settings
  - Inspiratory pressure
  - Inspiratory time
  - Expiratory time
  - Triggering sensitivity
  - Air-oxygen mix
- Monitors and Alarms
  - Patient disconnect (low pressure)

dom are all of these functions mentioned individually in ventilator orders, but all of them must be set by *someone* each time a patient is placed on the ventilator, and any one of them can substantially affect the patient's care.

Table 3. Ventilator Orders 1992: Components in the Siemens Servo 900

- Controls and Settings
  - Mode
  - Breaths/minute
  - Inspiratory time
  - Pause-time breath
  - SIMV breaths/minute
  - Low/high rate
  - Working pressure
  - PEEP
  - Inspiratory pressure level
  - Upper pressure limit
  - Inspiratory flow pattern
  - Triggering sensitivity
  - F_{IO_2} (O_2 mixer)
  - F_{IO_2} high-limit alarm
  - F_{IO_2} low-limit alarm
  - Minute ventilation high-limit alarm
  - Minute ventilation low-limit alarm
  - Adult/infant range
- Monitors and Alarms
  - Peak airway pressure
  - Minimum airway pressure
  - Mean airway pressure
  - Delivered volume/breath
  - Delivered volume/minute
  - Exhaled volume/breath
  - Exhaled volume/minute
  - Breaths/minute: Ventilator
  - Breaths/minute: Patient
  - Breaths/minute: Total
  - Percent O_2: Set
  - Percent O_2: Delivered
  - Electrical power supply
  - Gas supply

Who Should Decide?

In most patient care settings, it has traditionally been the physician who makes important management decisions. The physician decides whether to administer medication, selects the drug to be used, and determines the dose and schedule. In the past, it has usually been the same with ventilator orders. However, the increasing complexity of ventilators and their use, and the emergence of the respiratory care practitioner, a health-care teammember who is specifically trained in ventilator management, have dramatically changed this situation in the United States. Today, in many cases, the ordering physician is unfamiliar with the features of the ventilator being used on his or her patient, and even specialists in pulmonary and critical care medicine may not be comfortable adjusting all the items listed in Table 3. The respiratory care practitioner, on the other hand, knows the ventilator thoroughly, but may or may not have an appropriate understanding of its role in the patient's overall management.

What aspects of mechanical ventilation should be ordered by a physician, and which by a respiratory care practitioner or other nonphysician? Consider the following list:

Mode (ie, assist/control vs SIMV vs pressure control ventilation)
Full vs partial ventilatory support
PEEP
Inspiratory pressure
Tidal volume
F_{IO_2}
Inspiration-expiration ratio
Inspiratory pressure waveform
Alarms
Initiation of weaning
Extubation

Should these variables be chosen by a physician or by a respiratory therapist? If they should be decided only by a physician, then which physician? Often, patients are admitted by their personal physicians—general practitioners or specialists from a wide variety of disciplines—and seen in consultation by other physicians specializing in respiratory care-related fields. Should the latter be intensivists or pulmonologists or anesthesiologists? And, if respiratory care practitioners are to manage all or some aspects of ventilator care, what credentials should they have? Should they be registered (RRT), or certified (CRTT), or specially qualified in some other way? What about physician's assistants, nurses, physical therapists, and clinical engineers?

Should the person managing mechanical ventilation have direct clinical knowledge of the individual patient being ventilated? Should that person also have a thorough understanding of cardiorespiratory physiology and pathophysiology, plus a conceptual grasp of ventilation strategies, available modes, and ventilator capabilities? Most clinicians would agree that these things would be desirable, if not mandatory. But what about familiarity with every control and setting on the ventilator being used? And, for that matter, a working knowledge of the circuitry and mechanics of that ventilator? As desirable as these attributes may be, it becomes apparent that there may be too much for most individuals dealing with patients—whether physicians or respiratory care practitioners—to know about ventilator management.

One dictionary defines an order as "a command, direction, or instruction, usually backed by authority."[10] This definition emphasizes the fact that expertise cannot be the whole story in ordering mechanical ventilation. For numerous reasons, alluded to earlier and listed in Table 1, whoever manages mechanical ventilation needs the *authority* to do so, both professionally and legally. Who is granted this authority will vary a great deal in different hospitals, in different population centers, and in different parts of the world. Some of the factors that may affect how ventilator management is carried out in a particular institution are listed in Table 4.

Ventilator Settings or Outcomes?

An order for mechanical ventilation can take several forms. It may specify every possible setting and alarm, or it may be much more general and leave the details to someone other than the writer of the order. One may consider a ventilator order as occupying one of three possible levels (Table 5). Here the term level could refer to the level of expertise of the person writing the order, the level of sophistication of some second person whose actions are being ordered, or the level of independence granted to that individual in managing the patient.

Table 4. Who Should Order and/or Implement Mechanical Ventilation?: Institution-Specific Variables

- Hospital policy
- Medical staff policy
- Knowledge of physician
- Technical skills of physician
- Knowledge of respiratory care practitioner
- Technical skills of respiratory care practitioner
- Type and complexity of individual case
- Type and complexity of management
- Staffing and coverage considerations
  - Physician
  - Respiratory care practitioner
  - Nurses

One problem with Level-1 ventilator orders is illustrated in Table 6, which chronicles the ventilator orders for a particular patient in my institution during one 48-hour period. At this teaching hospital, only physicians (usually residents) can write ventilator orders, and features such as mode, tidal volume, F_{IO_2}, and cycling frequency can be changed only by specific order. Many orders are given at night, in response to a call from the patient's nurse, often by cross-covering residents not fully familiar with the patient, and at times when the advice of more senior trainees and attending physicians is less readily available.

Table 5. Possible Approaches to Ordering Mechanical Ventilation

Level 1:	Individual Ventilator Settings	
	Examples:	"Decrease PEEP from 15 to 10 cm H_2O" "Draw ABG in 30 min"
Level 2:	Outcomes	
	Examples:	"Adjust SIMV rate to keep pH 7.35-7.45" "Adjust F_{IO_2} to keep saturation 90-95%"
Level 3:	Protocols	
	Examples:	"PEEP trial per protocol" "Wean and extubate patient per protocol"

The orders in Table 6 reflect fine-tuning ventilator changes in a patient whose underlying condition changed little during the period in question but whose saturation and arterial blood gas values were

Table 6. Examples of "Level-1" Ventilator Orders*

"Decrease IMV rate to 14/min"
"Increase F_{IO_2} to 0.40"
"Increase IMV rate to 17/min"
"Decrease F_{IO_2} to 0.30"
"Increase IMV rate to 19/min"
"Decrease IMV rate to 16/min"
"Increase F_{IO_2} to 0.40"
"Decrease IMV rate to 14/min"
"Increase IMV rate to 16/min"
"Increase IMV rate to 18/min"

*Orders written on one patient at the author's institution during one 48-h period. The patient was clinically stable but experienced periods of agitation. At this institution, an arterial blood gas specimen can be drawn at the nurse's discretion without a physician's order; arterial blood is always drawn and analyzed after a change in ventilator settings.

fluctuating. For each fluctuation, the physician had to be notified and a new order given, to be followed up by further measurements after a suitable interval. The management of this patient would have been simplified if the ventilator orders had specified outcomes (eg, Level 2) rather than input data. If a range of permissible tidal volumes, pH and P_{CO_2} values, and F_{IO_2}s were specified, fine-tuning could be carried out by the respiratory care practitioners at the bedside, coordinated with the nursing staff, without direct input from the physician.

Protocol-Driven Ventilator Management

The third level of ventilator orders remains hypothetical for many institutions but reflects a growing trend within respiratory care that will surely find wider acceptance. Figure 1 illustrates the use of a therapist-driven protocol to determine appropriate technique for aerosol bronchodilator therapy at one hospital.[11] Such a protocol, once established by the respiratory care department, with input from its medical director and subsequent approval by the hospital's medical staff, allows the patient's physician to write an order for "aerosol bronchodilator protocol" rather than specifying the route, dose, and frequency of drug administration as is traditionally done. Within the limits allowed by the protocol, the respiratory care practitioner can assess the patient, determine the most appropriate and cost-effective delivery technique, and monitor the effects of therapy.

At the hospital in question, a second protocol is also used to determine whether aerosol bronchodilator therapy should be continued, thus eliminating unneeded therapy and improving the efficiency of the staff members involved.[11] At the same institution, protocols have also been used successfully for oxygen therapy (Fig. 2), including built-in provisions for contacting the patient's physician whenever unexpected abnormalities or an inadequate therapeutic response is encountered.[12]

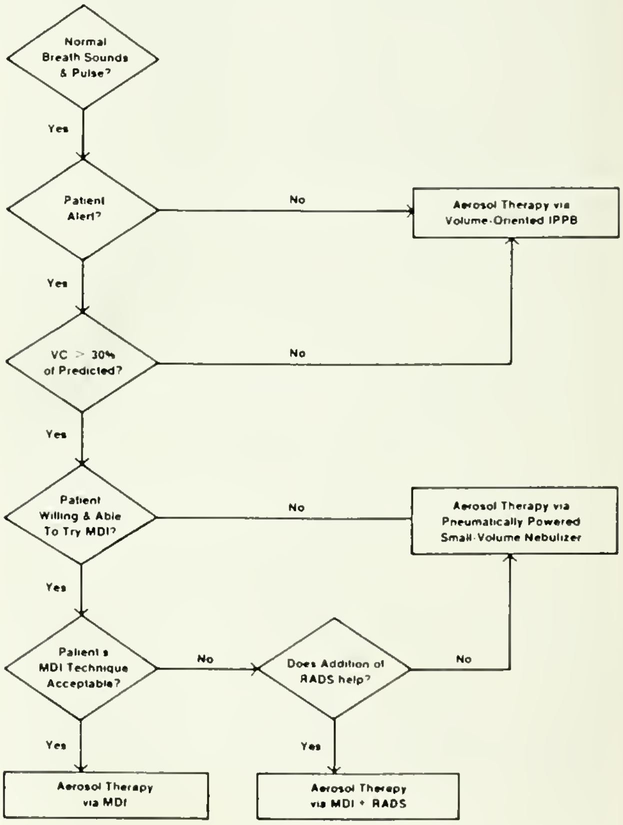
Normal Breath Sounds & Pulse?
Yes
Patient Alert?
No
Aerosol Therapy via Volume-Oriented IPPB
Yes
VC > 30% of Predicted?
No
Yes
Patient Willing & Able To Try MDI?
No
Aerosol Therapy via Pneumatically Powered Small-Volume Nebulizer
Yes
Patient's MDI Technique Acceptable?
No
Does Addition of RADS help?
No
Yes
Yes
Aerosol Therapy via MDI
Aerosol Therapy via MDI + RADS

Fig. 1. Example of an assessment protocol to determine appropriate technique for aerosol bronchodilator administration. The patient's physician orders the protocol, which permits the respiratory care practitioner to determine the appropriate technique according to guidelines specified in the protocol and agreed upon by the medical staff. A separate protocol is used by the respiratory care practitioner at specified intervals to determine whether the therapy should be continued. IPPB = intermittent positive-pressure breathing; VC = vital capacity; MDI = metered dose inhaler; RADS = reservoir aerosol delivery system. (Reproduced, with permission, from Reference 11.)

The therapist-driven protocol is well suited for ventilator management (Table 5), although at present there is less clinical experience in this setting than with the therapies described. At LDS Hospital in Salt Lake City, Utah, protocol-driven management of patients with severe acute respiratory failure has been used successfully for several years, relying heavily on a computer-based algorithmic protocol.[13,14] No doubt other institutions will report experience with this type of ventilator management in the coming years.

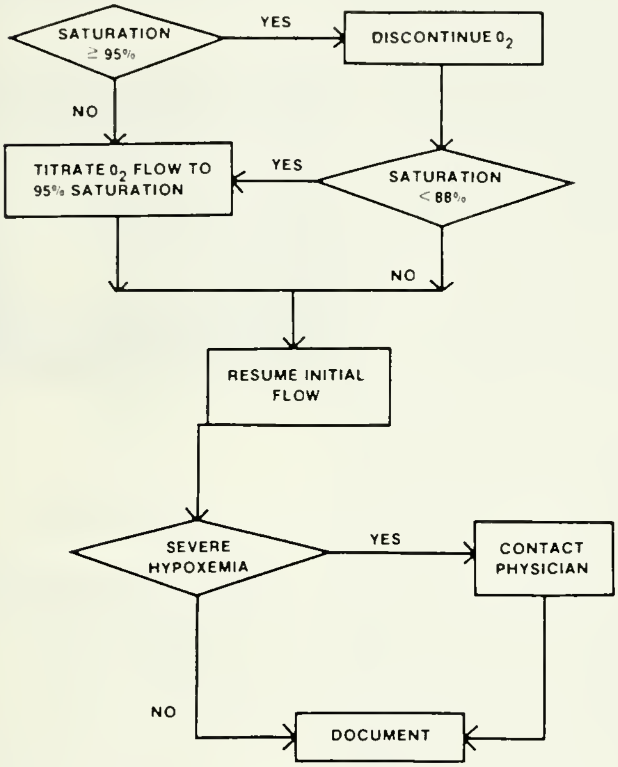

Fig. 2. Example of a therapist-driven protocol for oxygen therapy assessment and administration. Automatic physician contact and decision making are built into the protocol in the event of adverse event or unsuccessful therapy. (Reproduced, with permission, from Reference 12.)

Conclusions and Recommendations

Variations in local medical practice, hospital bylaws, availability of skilled respiratory care practitioners, and individual expertise all affect how ventilator orders are given in an individual institution. Whether ventilator management is ordered and carried out by specialist physicians, generalist physicians, respiratory care practitioners, or others depends on these and other factors. However, the requirements for effective, safe ventilator management are the same regardless of how such issues are handled at a particular hospital.

Based on the foregoing discussion and related considerations, I recommend the following with respect to ventilator orders:

- All ventilator orders should be part of the permanent hospital record and should be documented appropriately.
- A record of current ventilator orders and all current settings should be readily available in the ICU.
- Whenever possible, ventilator orders should take the form of specified outcomes (eg, pH, P_{O_2}, total rate) or initiation of an established protocol.
- An order for ventilatory support may authorize appropriate individuals (eg, qualified respiratory care practitioners) to independently adjust the degree and form of such support, in accordance with guidelines established for the clinical setting in which the order is given.
- Ventilatory support should be managed by someone who is competent in both its medical and its technical aspects, specifically:
  - initiation of ventilatory support
  - weaning and extubation
  - patient monitoring related to ventilatory support.
- Ventilatory support should be managed by someone who is competent in the selection and adjustment of
  - ventilatory mode
  - F_{IO_2}
  - inspiratory pressure
  - end-expiratory pressure
  - tidal volume
  - breath rate.

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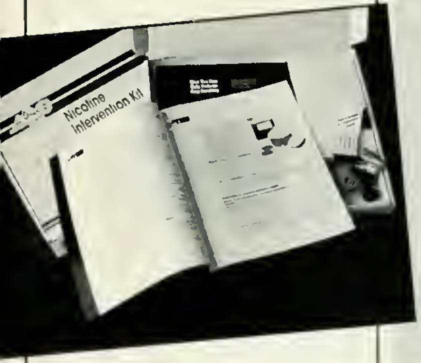

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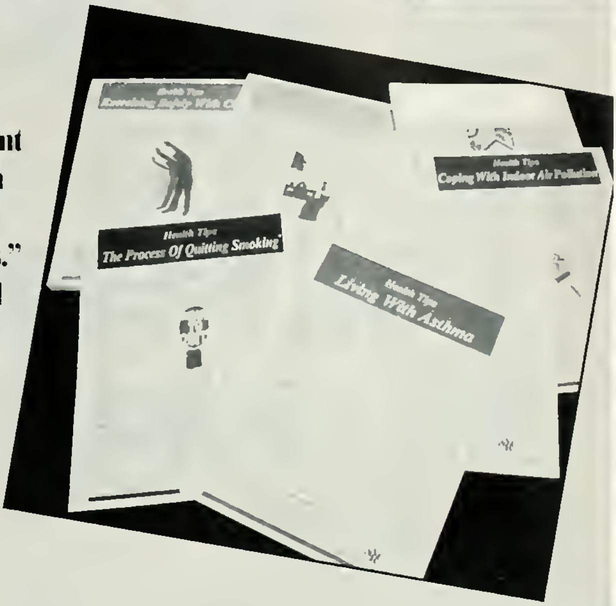
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AARC & AFFILIATES

September 17-18 in Tucson, Arizona. The AzSRC hosts its Annual Meeting at the Tucson National Resort, Golf Club, and Spa. Contact Becky Shocklee, 3341 N 31st St #108, Phoenix AZ 85016.

September 17-18 in Bethel, Maine. The Annual Fall Seminar, presented by the MSRC, is held at the Bethel Inn & Country Club. Come get a taste of New England and its beauty in the Fall. Enjoy activities such as an 18-hole championship golf course, tennis, canoeing, swimming, an outdoor barbecue, and much more! Plenty of outstanding speakers, topics, and vendors present the latest in available technolo-gies. Contact Edward Amend, 2810 Isthmus Rd, Rumford ME 04276-9724. (207) 364-4581, ext 362.

September 23 AARC Videoconference. The AARC, in conjunction with VHA Satellite Network, presents "Aerosol Administration," one in a series of live satellite videoconferences titled "Professor's Rounds in Respiratory Care." Featured presenters are David J Pierson MD and Dean Hess MEd RRT. Site registration for entire staff is $245 for AARC members. Call (214) 830-0061.

September 24-25 in Indianapolis, Indiana. The ISRC presents its Fall Conference at the Marriott Inn. The awards ceremony and Sputum Bowl are held on Thursday. Contact Jan Doherty (800) 327-3152 or (219) 761-3607.

September 24-25 in Napa, California. The CSRC, Chapter 10, the American Lung Association of the Redwood Empire, and the respiratory therapy program at Napa Valley College present the 10th Annual Napa Valley Conference, "Current Concepts in Cardiopulmonary Care." Topics include international strategies on asthma therapy, alternative methods of mechanical ventilation, liquid perfluorochemical ventilation, update on clinical practice guidelines, HIV infection in children, and reperfusion therapy. Speakers include Lana Hilling, CRTT, and Drs John Hodgkin, Irwin Ziment, James Hurst, and William Ganz. Call (707) 253-3145.

September 29-30 in Honolulu, Hawaii. The HSRC holds its 19th Annual Respiratory Care Conference at the Hilton Hawaiian Village Hotel. Contact Helen M Ono RRT, 1717 Palolo Ave, Honolulu HI 96816. (808) 547-9532.

September 30-October 2 in Traverse City, Michigan. The MSRC presents its Annual Fall Conference at the Shanty Creek/Schuss Mountain Resort. The general session focuses on asthma management, critical care, ventilation and waveform interpretation, nutrition, and risk management. Also, the Cardiopulmonary Diagnostics Membership Section presents pulmonary function and sleep disorder testing. Social events include a wine-and-cheese reception, golf outing, cookout, and dance. Contact Beth Hill RRT, Bay Medical Center, Respiratory Care Dept, 1900 Columbus Ave, Bay City MI 48708. (517) 894-3166.

October 2-4 in Key West, Florida. Chapter 1 of the FSRC and Sunset Seminars present the 6th Annual Southernmost Sunset Seminar at the Holiday Inn Beachside. Registration includes a Saturday evening conference aboard the Atlantic X dinner/casino cruise ship. Contact Dave Robbins, Coral Gables Hospital, 3100 Douglas Rd, Coral Gables FL 33134. (305) 441-6819.

October 14 in Auckland, New Zealand. The NZSRC presents its Annual Scientific Meeting at the Aotea Centre. The meeting precedes the Australian-New Zealand Intensive Care Society Conference. Contact Graeme A'Court, PO Box 10148, Balmoral, Auckland, New Zealand. (643) 640640.

October 16 in Long Island, New York. The Southeastern Chapter of the NYSSRC presents its 24th Annual Symposium, "Focus on Technology," at the Marriott Hotel in Uniondale. Speakers include Michael McPeck, Nathan Seriff, Joseph Lore, Steven Marzo, Margaret McGovern, and AARC President Bob Demers, who present "Clinical Hazards Associated with Aerosols." Contact Ken Nugent RRT (516) 444-8511.

October 28-29 in Sturbridge, Massachusetts. The MSRC presents its 15th Annual Meeting and Exhibition at the Sturbridge Host Hotel and Conference Center. Topics include adult and neonatal ventilator management, the RCP's role in smoking cessation, and interpretation of ventilator graphics. Events include Sputum Bowl, golf tournament, and awards banquet. Contact Bill McGarry CPFT RRT, Respiratory Care, Beth Israel Hospital, 330 Brookline Ave, Boston MA 02215. (617) 284-0782.

October 30 in Bear Mountain, New York. The Hudson Valley Chapter of the NYSSRC presents its Annual

Educational Seminar, "Challenging Issues in the Respiratory Care Environment," at the Bear Mountain Inn. Topics include patient assessment, infection control, and risk management. Contact Larry Lyman (914) 358-6200, pager 600.

February 16-19 in Reno, Nevada. The American Lung Association of Nevada and The Nevada Society for Respiratory Care presents the 12th Annual High Sierra Critical Care Conference at the Peppermill Hotel Casino. Topics include adult, pediatric and neonatal critical care issues. Contact Donna Turner, American Lung Association of Nevada, PO Box 7056, Reno NV 89510, (702) 829-5864 (9 am - 3 pm PST).

OTHER MEETINGS

September 17-20 in Chicago, Illinois. The University of Health Sciences/Chicago Medical School, Office of Continuing Medical Education, and the Institute for Critical Care Medicine sponsor the 11th Annual Chicago Critical Care Symposium and Review Course. The meeting will be held at the Hyatt Regency O'Hare. Contact Elizabeth Bruechert, CME Office, 3333 Green Bay Rd, N Chicago IL 60064, (708) 578-3214, fax (708) 578-3320.

September 18 in Beaumont, Texas. The respiratory care department of St Elizabeth Hospital presents its 4th Annual Educational Seminar. National and local speakers lecture on a wide variety of topics for practitioners of all specialty areas. Contact Greg Rodgers RRT (409) 899-7065.

September 21-22 in Ann Arbor, Michigan. The University of Michigan Medical School's Department of Internal Medicine presents "Update on Pulmonary and Critical Care Medicine." The course features updates on controversies and evolving technologies in pulmonary and critical care medicine. Contact Edwina Borde, Conference Registrar, Towsley Center for Continuing Medical Education, Department of Post Graduate Medicine, University of Michigan Medical School, PO Box 1157, Ann Arbor MI 48106-1157.

October 9-10 in Vail, Colorado. The Colorado Advanced Life Support Committee holds its Biennial Resuscitation Conference, "Fresh Perspectives on ACLS and PALS," at the Westin Hotel. Join Joseph Ornato MD and James Seidel MD, among others, for state-of-the-art therapy and a review of the Dallas ECC Conference during the beautiful Fall season in the Rockies. Contact Colorado ALS, PO Box 440895, Aurora CO 80044, (303) 363-8380.

October 11-13 in Arlington, Virginia. The first National Asthma Conference is scheduled to hold a workshop specifically for respiratory care practitioners during the conference. The workshop has been approved for 9 continuing education credits by the AARC, and is open to any respiratory care practitioner who wishes to attend. Contact NHLBI (301) 951-3275.

October 11-18 in Pulmonary Patient Cruise. LIFE Unlimited presents a "Western Caribbean Cruise" for pulmonary patients, their families, and friends aboard the S.S. Seabreeze. Cruise from Miami to Grand Cayman, Montego Bay, and Cozumel with private escorted tours and 24-hour professional services. Reservation deadline is Sept. 4. Contact Dave Robbins, LIFE Unlimited, 17101 SW 200 St, Box Z28, Miami FL 33187, or call (800) 621-4571 or (305) 441-6819.

October 24-29 in Jerusalem, Israel. XIV World Congress of Asthmalogy. Topics to be presented include molecular biology in lung disease, cells involved in asthma, and house dust mite and asthma. Abstracts are being accepted now. Contact Gil Kenes, 1617 JFD Blvd, Suite 946, Philadelphia PA 19103, (800) 223-3855.

November 18-21 in Miami Beach, Florida. The University of Miami, Division of Neonatology-Department of Pediatrics, presents its Annual Neonatal Symposium, "New Developments in Neonatal Respiratory Care," at the Doral Ocean Beach Resort. The symposium will include lectures, discussions, and a workshop on pulmonary function testing in neonates. Contact Charles R Bauer, MD, Program Co-Director, University of Miami-Department of Pediatrics (R-131), PO Box 016960, Miami FL 33101, (305) 547-5808, fax (305) 547-3501.

March 3-5 in Lyon, France. The Journées Internationales de Ventilation à Domicile presents the International Conference on Home Mechanical Ventilation. The meeting includes scientific sessions, practical education sessions, and poster presentations and exhibits. Simultaneous translation (French/English) will be offered. Deadline for abstracts is December 1992. Write: JIVD (SRMAR) 93, Grande Rue de la Croix-Rousse, 69317 Lyon Cedex 04, France, or call (33) 78 39 08 43, fax (33) 78 29 98 94.

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171	172	173	174	175	176	177	178	179	180	181	182	183	184	185
186	187	188	189	190	191	192	193	194	195	196	197	198	199	200

I. Type of Instn/Practice
1 ☐ Hosp 500 or more beds
2 ☐ Hosp 300 to 499 beds
3 ☐ Hosp 200 to 299 beds
4 ☐ Hosp 100 to 199 beds
5 ☐ Hosp <100 or less bedS
6 ☐ Skilled Nursing Facility
7 ☐ Home Care Practice
8 ☐ School
II. Department
A ☐ Respiratory Therapy
B ☐ Cardiopulmonary
C ☐ Anesthesia Service
D ☐ Emergency Dept
III. Specialty
1 ☐ Clinical Practice
2 ☐ Perinatal Pediatrics
3 ☐ Critical Care
4 ☐ Clinical Research
5 ☐ Pulmonary Function Lab
6 ☐ Home Care/Rehab
7 ☐ Education
8 ☐ Management
IV. Position
A ☐ Dept Head
B ☐ Chief Therapist
C ☐ Supervisor
D ☐ Staff Technician
E ☐ Staff Therapist
F ☐ Educator
G ☐ Medical Director
H ☐ Anesthesiologist
I ☐ Pulmonologist
J ☐ Other MD
K ☐ Nurse
V. Are you a member of the AARC?
1 ☐ Yes 2 ☐ No

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186	187	188	189	190	191	192	193	194	195	196	197	198	199	200

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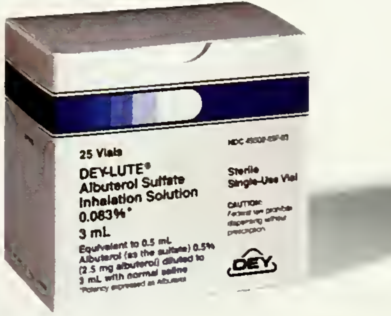

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